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OF

OPHTHALMOLOGY

EDITED IN ENGLISH AND GERMAN

BY

DR. H. KNAPP AND DR. C. SCHWEIGGER
OF NEW YORK OF BERLIN

DR. F. E. D'OENCH
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VOL. XIX. No. 1.

ARCHIVES OF OPHTHALMOLOGY.

EXTRA-PAPILLARY COLOBOMATA.

By G. LINDSAY JOHNSON, M.A., M.B., B.S., Contab.,
F.R.C.S., London.

OPHTHALMIC SURGEON WEST-END HOSPITAL FOR NERVOUS DISEASES ; OPHTHALMIC SURGEON WESTERN GENERAL DISPENSARY ; AND CLINICAL ASSISTANT MOORFIELDS EYE HOSPITAL.

(*With eleven wood-cuts and eight chromo-lithographic plates.*)

I HAVE chosen the name *extra-papillary colobomata* to signify all those colobomata which do not involve the optic disc, and which are therefore distinct from the classical coloboma due to the imperfect closure of the fœtal cleft, as well as the coloboma of the sheath of the optic nerve, so fully described by Fuchs, van Duyse, and others. I wish to avoid the term central or macula coloboma, because I think I am in a position to show that extra-papillary colobomata, identical with those described by previous writers as peculiar to the macula lutea, may be found in any part of the fundus whatsoever, and I think the reason why they have hitherto been found only about the macula and the disc is, that these are the two parts which form the chief features of interest in the fundus, and are therefore those most carefully examined. Moreover, colobomata unconnected with the disc or macula occur for the most part in absolutely healthy eyes possessing normal vision, and have consequently been overlooked. The so-called macula coloboma must, therefore, be included in, and considered merely a variety of, the extra-papillary colobomata I am about to describe.

Several writers (v. Ammon, Brücke, Huschke, Manz[1]) have tried to explain the occurrence of the macula coloboma as due to a rotation of the eyeball, and a consequent displacement of the fœtal cleft; but I am convinced that when I have mentioned some of the cases which have come to my notice, it will be clear that this theory must be abandoned. Hoffman has floated the ingenious theory that colobomata confined to the choroid may be due to intraocular pressure, tearing asunder the barely closed fœtal cleft; but here again, apart from *a priori* objections, many of my cases are incompatible with this hypothesis. Schweigger's theory,[1] which might be termed the "fœtal inflammation" theory, is very widely accepted at the present time, and overcomes many difficulties in the way of the other suppositions, and yet, although affording a probable clue to certain cases, will, I believe, fail in accounting for some of the cases I hope to show.

---

[1] "Ueber ein Colobom der inneren Augenhäute ohne Colob. d. Iris." Dissert, Frankf., 1871.

The first point which I think I can demonstrate is that extra-papillary colobomata can occur in any part of the fundus, and I believe that the number of cases which have already come to my notice are sufficient to authorize my making such statements.[2] Besides the colored drawings of twelve cases, published with this paper, I have in my possession a number of other drawings similar in character to these.

The two following diagrams represent the fundus of the right and left eye, and I have indicated thereon a number of the extra-papillary colobomata which I have seen, as well as some few which have been illustrated by previous writers. I may, however, add that those illustrated by previous writers are, with scarcely an exception, found in the region of the macula.

All the above cases, with five exceptions, have come under my own observation. Two of these exceptions, Nos. 4 and 20, are inserted by kind permission of Prof. Tweedy, and the three remaining are copied from the drawings of

---

[1] Schweigger: "Lehrb. d. Augenheilkunde."

[2] The same remarks apply equally well to iris-colobomata. The usual direction we know is downwards, and deviations from that direction were considered not to be colobomata (Manz: *Graefe-Saemisch. Hand-book*, II., 2, p. 65); but v. Mittelstädt has described cases occurring both outwards and inwards; and Prof. Tweedy assures me he has met with an undoubted congenital iris coloboma directed vertically upwards.

Diagrams showing the relative size and position of various colobomata that have come under my notice or have been elsewhere figured and described. To avoid confusion the very numerous "central" colobomata have, with two exceptions, been omitted. The ordinary "fissure" colobomata have also been left out.

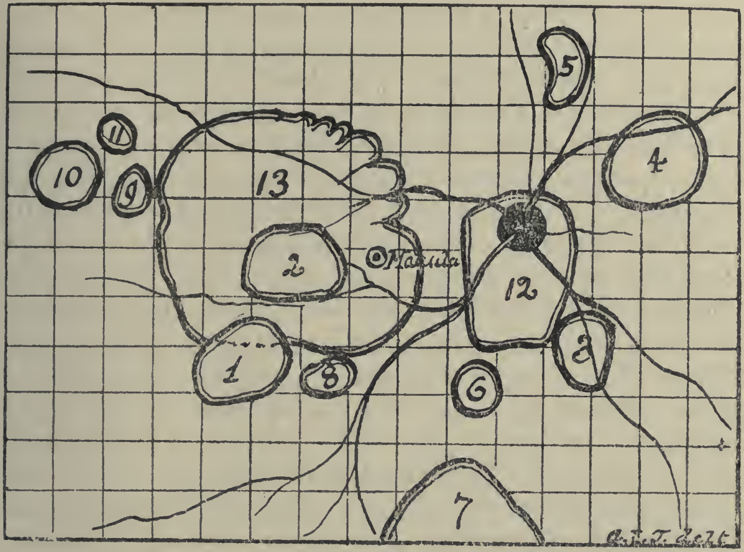

FIG. 1. R. EYE.

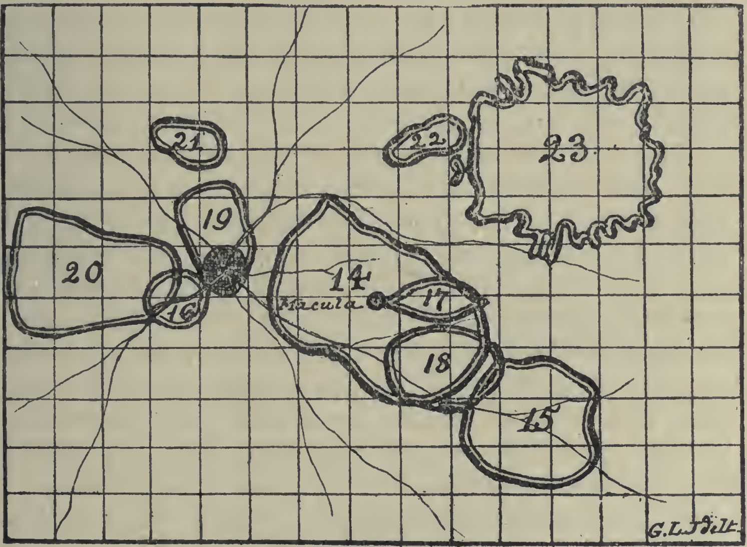

FIG. 2. L. EYE.

EXPLANATION OF DIAGRAMS.—FIG. 1.—R. EYE (erect image). 1. Pl. I., Fig. 1. 2. Pl. IV., 7. 3. Pl. V., 10. 4. Mr. Tweedy's 1st case. 5. John B——. An unpublished case drawn and described by me at Moorfields. 6, 7. Hirschberg's case figured in the *Centralbl. f. p. Augenh.*, Aug., 1885. 8, 9, 10, 11. The remarkable case of W—— S——. Pl. VII., 13. 12. Papillary coloboma. Pl. VI., 12. 13. Macula coloboma. Pl. VII., 13. FIG. 2.—L. EYE (erect image). 14. Macula coloboma. Pl. VIII., 14. 15. Extra-papillary coloboma. Pl. VIII., 14. 16. Schweinitz and Randall's case. A. OF O., Vol. xvii., Part 4. 17. Pl. V., 9. 18. Pl. II., 4. 19. Pl. IV., 8. 20. Mr. Tweedy's 2d case. 21, 22. A recent case. A large very characteristic "macula" coloboma was present in the same eye, and a papillary coloboma in the right eye. Unpublished. Found and described by me at Moorfields. 23. Fuchs' case. See ARCHIVES OF OPH., 1883, p. 37, with plate.

Prof. Hirschberg, Prof. Fuchs, and Dr. Randall. Only two central colobomata have been drawn (Nos. 13 and 14), which, being larger than any previously described, would probably include in their circumference all the rest. The typical "fissure" colobomata have likewise been omitted. The outlines of the colobomata are drawn accurately to scale, and to assist the reader in grasping their size the diagrams are divided into squares, each the size of the average optic disc. Some of the colobomata are very large. One has only to compare the small circle (No. 13)—de Schweinitz and Randall's case—with their original drawing in the ARCHIVES, to appreciate the distance from the macula or disc at which congenital gaps may be found. I have to thank Dr. Paul J. Sartain, of Philadelphia, for first calling my attention to the case of Sarah S——, who came to the Western General Dispensary complaining of blepharitis. Suspecting hyperopia he examined her refraction with the ophthalmoscope, and this led to the discovery of the coloboma.

Prof. Tweedy's first case was discovered in a still more curious way. A man fell off the raised platform of one of our railway stations in attempting to turn round as he was walking with the railway line, on his left hand, and on recovering from the shock, thought he had injured his right eye. This resulted in an action for damages against the railway company, the plea being that the imperfect manner in which the station was lighted had contributed to his fall. and set up "choroiditis" in the eye. In the evidence, Mr. Tweedy stated that so far from the accident having caused the patch of "choroiditis," the so-called choroiditis had been the cause of the accident, it having evidently existed from birth, and, being situated upwards and inwards to the disc, had, owing to the scotoma it produced, prevented his seeing the edge of the platform, which was below and to his outer side. This will, I think, show that a careful study of these defects may have a practical issue.

Mr. Tweedy's second case, that of Rudolph D., æt, twenty, was examined ten years ago (1879), and then pronounced by him (in direct opposition to the opinion of a very eminent London oculist, who maintained that "the back of the eye was undergoing degeneration") to be a congenital malformation. Mr. Tweedy's last notes of the case two years ago show that no change whatever has taken place, the vision with + 1 D still being = $\frac{6}{6}$. In this case the remnants of a hyaloid artery, in the form of a white fluffy cord, could be seen stretching from the disc to the posterior capsule. A coloboma of the optic-nerve sheath was also present in this eye; the other eye was slightly myopic, R V with — 1 D = $\frac{6}{6}$, and showed no traces of any other congenital defect, except that the outer third of the disc was pigmented.

Nos. 21, 22 occurred in a girl, æt. twenty-two, who came to Moorfields for spectacles, as a patient of Mr. Lawson. Not getting more than $\frac{6}{18}$ with any glass, I examined the fundus, and discovered three characteristic punched out colobomata, one 3 P D's oval horizontally at the macula, one upwards and 3 P D's to the inner side of the disc, and one 3 P D's upwards and 3 P D's to the outer side of the disc. Except for a myopic crescent, and some shrinking of tissues on the outer side of the disc, there were no other defects.

I take it for granted that the reader will admit, in looking at the above diagrams, that there is reason for concluding that colobomata may occur in *any* part of the fundus, although the favorite seats are undoubtedly the region of the macula lutea and the choroidal fissure.

Reasons for the Macula Being a Favorite Seat.—Although one would naturally expect to find a coloboma remaining to bear witness to an imperfectly closed choroidal fissure, the reason why the macula should be so frequently the the seat is at first sight more difficult to account for; since von Ammon's rotation theory, as we have above said, only creates fresh difficulties. Many ophthalmologists, recognizing these objections, have endeavored to explain the macula colobomata on the hypothesis of intra-uterine choroiditis.

I think it unnecessary to go out of our way to seek the explanation in a disease which after all is only a presumption, and about which we know next to nothing, when we can find some facts to assist us close at hand. In the first place, the region of the macula is by nature a weak one. Hannover, in his "Memoir on the Funiculus Scleroticæ," says: "I have come to the conclusion that the fovea (centralis) is not only a formation due to an arrest of development, but also the most defective part of the entire retina (det mest defekte sted i hele Nethinden)."[1] According to him, this funnel-shaped cicatrix is situated in the sclerotic, immediately behind the fovea centralis, passing through the entire thickness of the sclerotic, and ending freely outside the globe in a kind of tag or short cord. He says he has examined 50–60 eyes, and has never found it absent. "One can also find traces of the fœtal cleft in the other portions of the sclerotic in the neighborhood of the funiculus . . . and these characters are still more pronounced in the eyes of the newly-born." He adds: "In the coloboma of the eye I have shown that the deep oval pit seen in the inside of the sclerotic behind the fovea, and the thinness of the membrane at this point, are due to an arrest of the (fœtal) cleft of the eye." It is not difficult to believe that in the formation of the secondary optic vesicle, this weak spot at the macula would be more likely to give way than any other part of the retina. Van Duyse even goes so far as to place the macula and the optic nerve in the middle of the embryonic fissure, and believes that in a normal eye the fissure to the outside of the optic nerve gets separated off from the embryonic fissure by the interposition of retinal elements.[2] I think this supposition lacks confirmation, and is certainly difficult to reconcile with Würzburg's interesting discovery of an embryonic macula lutea on the inner as well as the outer side of the papilla,[3] and still more so with the changes which these symmetrically placed yellow spots subsequently undergo.

In the second place, traces of arrested development apart from the coloboma may be seen in the great majority of cases. Thus, out of sixteen consecutive cases which I have met with, three of them showed traces of a hyaloid artery (see Plate III., Fig. 5); in four cases the eye was distinctly smaller than its fellow (see Plate II., Fig. 3; Plate VI., Fig. 11). In considerably more than half the cases the retina and choroid appeared thinned, or atrophied, round the margin of the disc, sometimes amounting to a distinct coloboma of the nerve sheath. Plate I., Fig. 2; Plate II., Fig. 4; Plate V., Fig. 9; Plate VII. and Plate VIII. The same may be observed among the cases described by Streatfeild, Randall, Montmeja, de Wecker, several of Van Duyse's Fuchs, Jessop, Tweedy, Hirschberg (1st case), Schnabel, Reich, and many others. Moreover, I noticed opaque, or at least translucent, nerve fibres in four cases. (Plate III., Figs. 5 and 6; Plate IV., Fig. 7.) See also van Duyse's case, *Annal. d'Ocul.*, No. 94. And in two cases (Plates VII. and VIII.) the whole outer portion of the disc included between the art. temp. sup. and art. temp. inf. was occupied by a network of choroidal veins, while the retinal pigment was more or less deficient right up to the coloboma.

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[1] Funiculus Scleroticæ en Levning af den fœtale Spalte i Menneskets Oie. Efterviist af Adolph Hannover. Meddelt i Videnskabernes Selskabs Mode d. 8. Dec., 1876.

[2] Du Colobome Central; *Ann. d'Oculistique*, Jan., 1884, p. 32.

[3] On the Development of the Eye in Mammals. A. OF O., 1887, p. 230.

It is curious that in no case of extra-papillary coloboma have I noticed any deficiency or change in the iris. Remak accounts for this fact on Hoffman's supposition that the intra-ocular pressure had torn asunder the barely closed fœtal cleft.[1]

Refraction.—Most of the cases were hypermetropic. Out of my sixteen consecutive cases three were emmetropic, three myopic (viz, 12 D, — 7 D, and — 9 D), eight hypermetropic, and two had mixed astigmatism. Now a small or undeveloped eye is usually associated with hypermetropia, and, consequently, this is what one would expect in colobomata of *small dimensions*. On the other hand, in *extensive* colobomata the coats of the eyeball would be seriously weakened, and tend to stretch and become staphylomatous, and thus directly induce a myopia, and that is what I found to be the case.[2] See Plates VII. and VIII. and Plate I., Fig. 2.

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[1] Ein Fall v. Colob. der Mac. lutea; *Centrabl.*, Sept., 1884.

[2] I think it must have been this latter class of cases which Schnabel had in his mind when he traced the connection between the etiology of myopia and the so-called coloboma circa maculam. See Schmidt-Rimpler, *Graefe's Archiv*, vol. xxvi., 2, p. 221.

And moreover the degree of myopia would be in proportion to the proximity of the coloboma to the posterior pole of the eye. Hence the myopia is considerable in the cases shown in Plate VII. and Plate VIII., while absent or nearly so in Fuchs' case, (see diagram L. E., No. 23). On the other hand, if the coloboma is of moderate size, *i. e.*, 1 to 3 P D's only, it may not affect the refraction at all.

Field of Vision.—The importance of ascertaining the exact sensibility to light over the coloboma area is obvious. Schmidt-Rimpler points out that it is not sufficient in order to establish a coloboma that one should have found a scotoma by means of the ball (or flag), but that every sensation of light, whether from the lamp or ophthalmoscope mirror, must be absent.[1]

Finding the signal made of white paper, commonly used, reflected far too feeble a light to be of decisive value in such an experiment, I constructed a small electric lamp for the purpose. See sketch opposite.

If these eight charts (which are samples of the rest) be examined we shall notice several facts:

1. The electric-light area for the whole field is slightly larger than the area for the white flag (reflected light) (see Charts 1, 3, 4, 6, etc.).

2. The electric-light area of the scotoma, though in every case smaller than with the flag test, is nevertheless quite definite, and shows that although the marginal retina (the pigment area) round the coloboma is sensible to the incandescent light, it is insensible to the stimulus of the reflection from the white flag (see Charts 1, 3, and 4).

3. Inside this zone the field is insensible to the most powerful stimulus.

4. In many cases the V F is very limited, and does not include the scotoma in its area, and yet a well-marked notch in the boundary line exists, both for the lamp and the flag in the region of the field corresponding to the coloboma, but somewhat nearer the axis of vision (Charts 1 and 2).

5. In one case no scotoma for the flag existed, because

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[1] *Augenheilkunde*, 3te Auflage, 1888, p. 236.

the section of the field in which the scotoma should lie was contracted within its inner boundary, and yet by the enlarged field given by the electric light the scotoma could be clearly brought out (see Chart No. 6). Compare also Chart No. 5, where scotoma is unseen for red and green, but brought out by white flag.

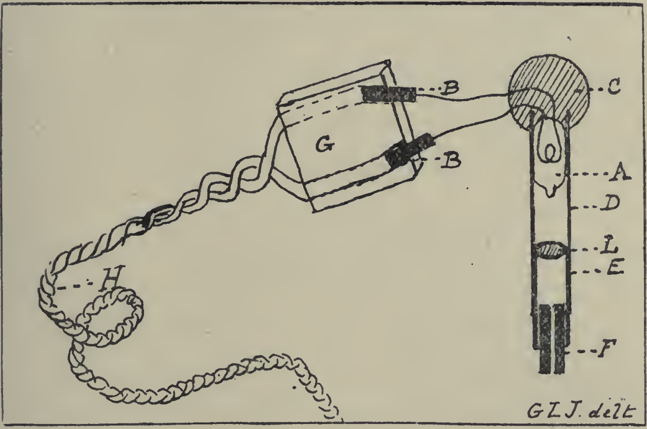

FIG. 3.

Fig. 3. Incandescent condensing lamp, used for ascertaining the area of partial and total insensibility to light in colobomata.

A is a very small incandescent lamp, which is fixed in position at the end of the brass tube D by means of a lump of plaster-of-Paris. This lamp is connected with the double wire H from the battery by means of two small wires which end in the short rods B B, fitting the two brass tubes in the block of vulcanite G. A second tube E slides inside the tube D, and at its end is fitted a biconvex lens of $\frac{3}{4}$ inch focus. By sliding this inner tube the lens may be placed exactly at its solar focal length away from the light A, and thus render all the emergent rays parallel. In order to limit the area of light still more, I drilled a 1 *mm* hole lengthways through a short rod of brass F, which fitted the second tube, and blacked the inside. To use the lamp the tube D slides into a fourth tube, which is soldered on to the runner which slides along the arm of the perimeter. This soldered tube was split along its length to admit the lamp tube easily, and was carefully directed, so that in every position along the arm the tube is in a line with the pupil of the patient's eye. The lamp is of two-candle power, and is worked by a storage battery of four volts. I have had it in use for several months and find it works admirably.

Lastly, very few of the scotomata corresponded in area or shape with the colobomata, as seen with the ophthalmoscope. This, I admit, is directly contrary to the observations of Schmidt-Rimpler,[1] De Wecker,[2] and others, but I made

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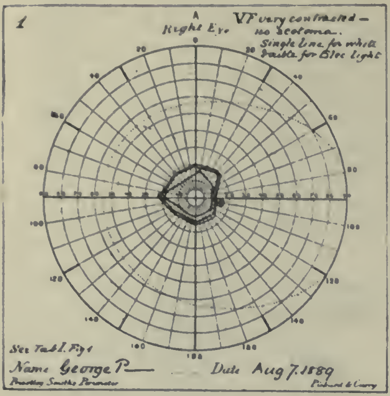
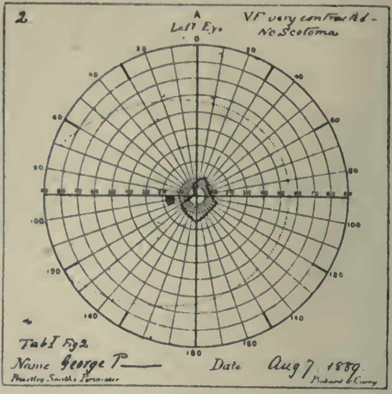
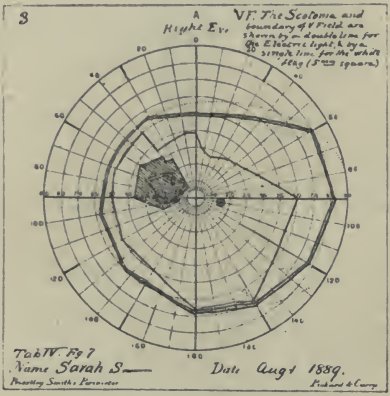
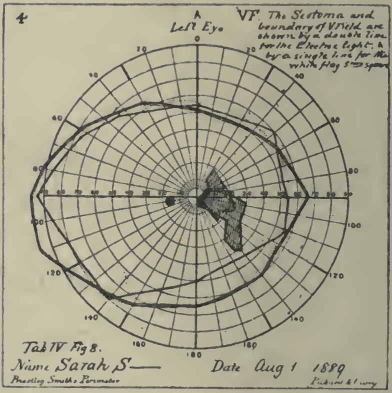
[1] *Graefe's Archiv*, xxiii., 4, p. 176.

[2] De Wecker et Landolt: "Traité Complet d'Oph.," vol. ii., p. 515.





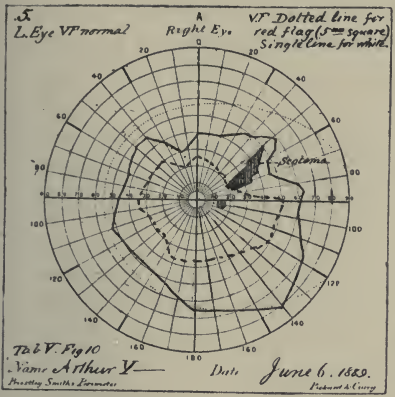


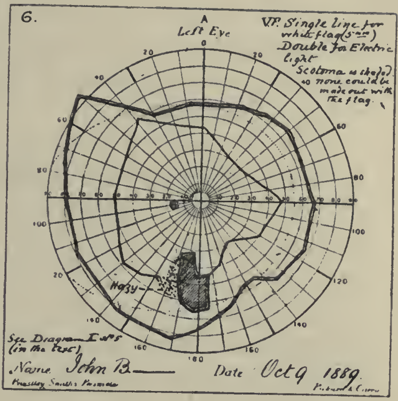


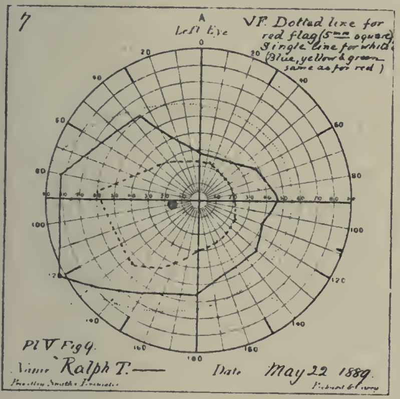
my observations so carefully that I have no doubt as to the correctness of the results.

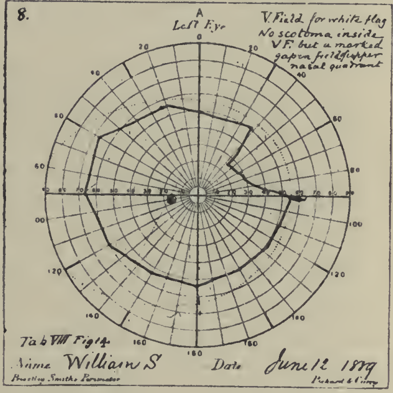
Charts 3, 4, and 6 are the only ones out of over thirty in which the scotomata even approximately resemble the shape of the coloboma, and that only held good for the electric light; white and red, green and yellow, giving shapes not only several times larger, but bearing no exact relation in position or shape to the coloboma as seen with the ope. directly.

Diagnosis.—It now becomes necessary to determine in what manner extra-papillary colobomata can be diagnosed so as not to be mistaken for cases of choroiditis and other inflammatory conditions of former and recent dates. Now,









the only diseases which could possibly be mistaken for as coloboma are:

A. Sclero-choroiditis posterior.

B. The plastic (exudative) group of choroidal affections, especially in their later (atrophic) stages. Under this head we include the disseminate varieties, both simple and specific, central, senile, and areolar choroiditis.

C. Subretinal and choroidal hemorrhages in their later stages.

It is beyond the scope of this paper to describe the above diseases, but I wish to point out the most salient characteristics which can guide us in diagnosing extra-papillary colobomata.

1. The margin of an extra-papillary coloboma is always sharply defined, and it is everywhere surrounded by healthy tissue, while it stands to reason that in disease this can rarely be the case.

2. In all cases of extra-papillary coloboma the pigment is found in front of the retinal vessels, and never behind, whilst in the above-mentioned diseases the pigment is not necessarily confined to any layer.

3. In choroiditis the tendency is towards the formation of multiple lesions, except in those forms such as senile and central choroiditis, which, however, are easily distinguished by other characters.

4. During the active stage of disease the appearance of the lesion naturally alters, whilst a coloboma always remains the same.

5. The most important characteristic of all, however, is the appearance and position of what I may be allowed to term the floor of the lesion. In coloboma one sees the sclerotic of a dazzling whiteness, forming the floor of a well-defined punched out pit, sometimes covered with a layer of connective tissue in a mother-of-pearl-like circular patch. In most cases of choroiditis the floor is of a dirty yellow color, or if chalky white other characteristics, such as the plurality of the lesions, and especially their small size will prevent any mistake.

Moreover, in choroiditis the depression, if any, is much less marked, the lesion often fading insensibly into the surrounding tissue, and often without any marginal layer of pigment. It has been pointed out, I think by Schnabel, that the margin of a coloboma is bounded by a whitish zone of atrophied tissue, and when seen is quite characteristic. I have certainly noticed it in some of my cases, but by no means in all.

I must here repeat that I am only dealing with extra-papillary colobomata, being aware how difficult it is to distinguish many colobomata of the optic nerve sheath from myopic crescents in connection with Sclero-choroiditis posterior.

In connection with the position of the floor, I would remark that, in *all* those cases in which the vision was normal and in some of the cases where it was deficient, I have found the difference of the focus between the margin of the coloboma and the floor amounted to between 2 D and 3 D.

By Dr. Landolt's tables for axial ametropia, based on the well-known formula $\vee - \varphi = \frac{\phi \phi'}{\mu - \phi}$, we find, by placing a lens of — 1 D at the anterior principal focus of an emmetropic eye, that the image is removed to a point 0.321 *mm* behind the retina. From the table of eye measurements in Quain's Anatomy we find the thickness of the sclera, choroid, and retina together = 0.08 in., and of the sclerotic alone = 0.05 in., giving for choroid and retina together a thickness of 0.03 in. = 0.75 *mm*. Now, if 0.321 *mm* corresponds to a correcting lens of — 1 D, a plane removed 0.75 *mm* farther back will need a concave lens of 2.5 D placed at the anterior focus.[1] This closely coincides with the actual lens used in the ophthalmoscope to bring the floor of the coloboma into focus. Of course, these figures are only to be considered approximately true, as we find it impossible to focus the fundus accurately within half a dioptry, and, moreover, it is open to doubt whether the thickness of the retina ought to be taken into account at all; but, making every allowance, I think it shows that in many cases of coloboma the depression was not an ectasia or staphyloma, but was *due to the absence of retina and choroid*, enabling us to see the sclerotic in its unaltered position.

Vision.—If the reader takes the trouble to glance over the notes of the cases appended he cannot but be struck with the excellence of the vision in many of the cases. Thus, in the case of Sarah S—— (Figs. 7 and 8, Plate IV.) and of Ralph T—— (Fig. 10, Plate V.) the vision was above normal = $\frac{6}{5}$ in both eyes, while in that of George P—— (Fig. 1) and George G—— (Fig. 3), as well as in Professor Tweedy's two cases (see diagrams), the vision = $\frac{6}{6}$. In Arthur Y——'s case the vision was nearly $\frac{6}{6}$. This was remarkable, as the coloboma encroached on the fovea itself, while in the case of Sarah S—— the coloboma were very near the fovea. I think such cases would argue strongly in favor of a congenital defect, as I have never known of a case of choroiditis close to the macula in which the vision was at or above normal.[1] I have not noticed any alteration in vision unless the coloboma actually covered the macula region.

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[1] The anterior principal focus is situated 13.7 *mm* in front of the cornea, which is as nearly as possible the position of the correcting lens of the ophthalmoscope when used in the direct examination of the fundus.

I might supplement my remarks under this heading of diagnosis by a reminder that, although we must not expect to find any pathological changes in a coloboma, yet the mere fact of the presence of a defect is sufficient to render the particular portion of the fundus more liable to be attacked by disease. To illustrate this I have inserted two drawings (Plate VII., 5 and 6), in which pathological changes in the neighborhood of the colobomata are present.

Pigment and Nævi.—We find in extra-papillary colobomata, as in many other cases in which the retina and choroid are atrophied, an agglomeration of pigment round the margin, and this pigment, moreover, is frequently distributed over the coloboma in the form of a veil or network which lies above the retinal vessels. In some cases the pigment merely forms a single ring bordering the coloboma; in others it forms a double contour or crescent, separated by an intermediate zone of thinned or atrophied choroid (see Figs. 1, 4, 6, 7, 8, 14). In others, again, the marginal pigment passes in the form of trabeculæ towards the centre of the coloboma, where it forms a fine veil or network. In many cases these trabeculæ are wanting, but their course can still be indicated by a few short processes or threads, as if the trabeculæ had been broken off and absorbed (see Figs. 9 and 14). These remains can be detected in nearly all cases in which the central network is absent, and I have seen them in many of the drawings made by previous writers, although their import has, to my knowledge, never been pointed out.

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[1] Mr. Nettleship, however, describes and figures a case of severe central choroiditis with a vision of $\frac{20}{20}$ nearly. But he adds: "Such changes involving, as they appear to do in this case, the fovea centralis, would be incompatible with such excellent vision as this eye possesses."—Trans. Oph. Soc., vol. v., p. 147.

Under this layer of pigment portions of the choroid are seen in an altered form. The choroidal vessels which pass into view from beneath the free edge of the coloboma unite together to form a dense impenetrable plexus towards the centre of the coloboma, which bears the closest resemblance to nævi seen elsewhere (see Plate I., Fig. 2, Plate V., Fig. 10, Plate VII. and Plate VIII). Nævi of the choroid and other tunics of the eye are not altogether unknown. Thus Lawford[1] gives a drawing of a microscopic section of the choroid in which there are unmistakable signs of a nævus, and he concludes that, "*a priori*, one feels inclined to say that the choroid, with its unusually vascular structure, would be a not unlikely situation for the development of a nævus." Horrocks[2] describes a case in which a port-wine colored nævus in the face was observed along with a nævus in the sclerotic and cornea, while the retinal vessels are extremely tortuous. Dr. Sturge[3] describes a case in which a congenital port-wine mark on one side of the face coincided with tortuous retinal vessels, and changes in the choroid on the same side. Lastly, Dr. Milles[4] mentions a case of nævus of the choroid accompanied by a detachment of the retina. The face was also marked by a large nævus.

Conclusion.—The cases published herewith will, I believe, already suffice to demonstrate that extra-papillary colobomata are not of as rare an occurrence as have been hitherto supposed, and I may add that whilst writing this paper I have observed several other cases, which only confirm what I have herein stated. I believe it will be found that they are not difficult to diagnose, and that with the exception of those cases which include the macula they do not impair the vision beyond causing a limitation of the field (unless they are so large as to weaken the resistance of the globe).

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[1] Trans. Oph. Soc., vol. v., p. 138.

[2] Trans. Oph. Soc., vol. iii., p. 106. See also Perrin and Poncet's "Atlas of Path. Anat. of the Eye," where a case described by them as "Congestion of Choroid" is believed by Dr. Lawford to have been a nævus.

[3] Clin. Soc. Trans., vol. xii., 1879, p. 162.

[4] Trans. Oph. Soc., vol. iv., p. 168.

If I may suggest some explanation of this evidently congenital defect, I think, as I have above indicated, that we are in the presence of something which presents many points in common with cutaneous nævi.

First.—The eye, although a highly differentiated nerve end-organ, is nevertheless essentially a skin formation, while the choroid presents an analogy to the corium both from its position, from its mesoblastic development, and from its affording nourishment to a directly superimposed layer of epiblastic pigment cells.

Secondly.—Wherever an agglomeration of choroidal vessels exists, we find it below the retinal pigment layer, which forms a network over it.

Thirdly.—The vessels forming the agglomerations are so fused together as to be indistinguishable and completely hide the sclerotic beneath.

Fourthly.—The absence of this dense structure in some of the cases can be explained by the analogous occurrence of the altered condition of the skin where nævi have disappeared and become absorbed, and I think it is not altogether unreasonable to suppose that the mother-of-pearl-like sheen, and peculiar glistening tissue which may be seen spreading over the base of some of the colobomata where this vascular mass is absent, is merely the connective-tissue cicatrix of an atrophied nævus.

Fifthly.—Cutaneous nævi are universally admitted to be congenital formations, and where they occur we find an increase of the pigment cells over them. Colobomata are likewise recognized as congenital formations, and the extra-papillary forms, at least, are conspicuous by the pigment clusters which surround and cover them.

Even had ocular nævi never been recorded, I hardly think it would be possible to come to any other conclusion, after examining the cases which form the subject of this paper, than that the peculiar choroidal structure in the centre of certain of these colobomata is of the nature of a nævus.

Lastly, it seems difficult to explain most of these malformations in any other manner, as *in no case could any history be obtained, either directly or indirectly, of syphilis; nor could*

I in any case (except perhaps that of George P——, Plate I.) *obtain the slightest evidence that any inflammation had occurred inside the womb during pregnancy, or that the mother had had any fever whatever.*

Moreover, the lesions have all the appearance of having a congenital origin, and, occurring as they do in every part of the field, cannot possibly arise from any fœtal cleft; besides, the well-known papillary colobomata, whether limited to the sheath of the optic nerve or passing down toward the iris, differ as a rule very materially in structure from the extra-papillary colobomata which form the subject of this paper.

CASE I.—George P., private patient, schoolboy, aged eight, youngest of four children, all healthy. Patient somewhat stunted in growth; head slightly hydrocephalic. Teeth fairly developed, enamel good, no notches. Hair light flaxen, approaching white. Irides blue. Excitable, delicate, subject to headaches. No history of syphilis in parents, nor any signs of it in patient. Right eye well developed; iris normal, active; lens normal. R V = $\frac{6}{6}$ nearly and J 1 at 12 in. No hypermetropia. Ophthalmoscope (see Fig. 1, Plate I.): fundus healthy; choroidal vessels invisible; disc pink, oval; retinal veins full, tortuous near the disc; arteries slightly tortuous in places; Y S small and granular, with well-marked "comet flare-spot."

Six P D's to the outer side, and 3 P D's below the papilla, is a large chalk-white gap, $2\frac{3}{4} \times 2$ P D's, surrounded by a pigmentary zone. This patch is exceedingly white, with a glistening mother-of-pearl sheen; over its surface a faint grayish sprinkling of pigment may be seen, but it is otherwise destitute of pigment or blood-vessels. Three small arteries and several veins can be traced to the patch, on reaching the edge of which they curl round and disappear, as in a glaucomatous disc, but, unlike the latter, they do not appear again. The edge of the white patch is abrupt and very sharply defined, and directly continuous with the surrounding retina and choroid of the fundus. The patch itself is distinctly on a lower level, which may be seen by the obvious parallax, and also by the difference of refraction, — 2 D, or 2.5 D by the direct examination. At the margin the retina advances beyond the choroid, in the form of a crescent-shaped lip, $\frac{1}{8}$ to $\frac{1}{12}$ P D in breadth, and is loaded with black-colored irregularly disposed pigment. The above-mentioned retinal vessels curl over the outer and thicker lip, and, apparently piercing the deep layers of the retina, run backwards between it and the choroid (*vide* Fig. 1, Plate I.). V F contracted to 20° nasal side, 15° temporal, 20° above, and 10° below; same for electric light, white flag, and colors.

Case 2.—Left eye of above patient, $V = \frac{6}{36}$, J 15 at 6″; sees slightly better at a distance with + 1.25 D. Sight has been defective from birth. The globe is slightly smaller than the right eye.

Ophthalmoscopic appearance (Fig. 2, Plate I.): The disc is slightly smaller than its fellow, round-oval, margin irregular, with faint pigmentary border. The triangle between the A. T. sup. and A. T. inf. is decidedly pale. The outer edge of the disc is bordered by a crescent, which fades imperceptibly into the thinned choroid. This latter forms a considerable patch, extending to at least half-way between the disc and the coloboma, about to be described. Owing to the more or less complete absence of the retinal pigment layer over this patch, the choroidal vessels are very conspicuous. The rest of the fundus (if we omit the coloboma) is quite normal, nor are the choroidal vessels visible anywhere else. At 2½ P D's from the edge of the disc is a large transversely oval-shaped patch, 4 P D's × 3 P D's. This presents some very characteristic points. The sharp bright-red margin of retina and choroid, which can be traced all round the coloboma, is covered with a lacework of dense black pigment, which forms an overlapping fringe. This border is irregular, forming ten well-defined notches filled up by black pigment, and from which as many thick processes or trabeculæ pass towards the central red mass. This latter presents the appearance of a nævus in structure, being evidently largely if not entirely formed of a dense anastomosis of choroidal veins and capillaries. These, for the most part, enter the coloboma beneath the pigment trabeculæ, and, following their course, form the above-described tangled mass. So dense is it that no trace of sclerotic can be seen between the meshes. The pigmentary trabeculæ also join to form an irregular lacework covering the nævus, which can be seen through the interstices, about 1 D below. These lobules, which are mapped out by the trabeculæ, and which bear a rough resemblance to the lobules of the cortex in a lymphatic gland, are, but for a stray retinal pigment and a few choroidal vessels, quite empty, and only show the bare glistering sclerotic shining through. The Y S is evidently absent, being displaced by the coloboma.

V F very contracted for electric light, 10° above, 20° below, 8° temp., 15° nasal. For white flag same-sized field, but different-shaped; slight gap outer side; no scotoma. See chart No. 2.

CASE 3 (Figs. 3 and 4, Plate II.).—George G——, aged three and one half. Kindly sent by Dr. de Carey. Patient is a bright and intelligent child, born at full time. Mother perfectly healthy, both before and during confinement. Patient is the youngest of four children—all healthy and have good sight. No trace of syphilis in the family.

Right eye, H 1 D; V = $\frac{20}{20}$ probably [owing to his early age (three and one half years), it was very difficult to get him to answer questions, but several letters of the test line were pronounced]. Disc, fundus, and Y S all healthy. Hyperopic reflex and halo round Y S.

Left eye, barely P. L.; globe undeveloped; cornea about $\frac{2}{3}$ diameter of right eye; refraction with ophthalmoscope and retinoscopy about + 1 D; lens well developed; no sign of cataract; iris blue, acts well to light—entire (Figs. 3 and 4, Plate II.) ophthalmoscope disc lost in a white or faintly pink egg-shaped coloboma 3 P D's × 2 P D's, long axis vertical. The four superior vessels terminate abruptly about the middle of the coloboma. The (numerous) lower ones curl over the lower edge of the coloboma, and, with one exception, disappear. At the place where the vessels curl over is a round glistening mother-of-pearl-like depression, nearly the size of a normal disc, at a somewhat lower level than the rest of the coloboma. The upper part of the coloboma is on a level with the general fundus, the choroid and retina of which do not pass quite up to the edge of the coloboma, but are separated everywhere by a white marginal ring destitute of choroid. A little pigment (retinal) is heaped up here and there round the coloboma. At $1\frac{1}{2}$ P D's external to this coloboma is a boat-shaped choroidal gap or coloboma $2\frac{1}{2}$ P D's × 1 P D, long axis horizontal. This is surrounded by a ring of black pigment and below by a second yellow ring of thinned choroid. The margin is strewn for some distance with fine pigment granules as well. Several choroidal vessels cover the gap from above inwards, and pass out of sight beneath the margin of the gap; the floor of the coloboma is a dirty white and is brought into focus with a — 2.5 D lens behind the mirror (direct); the choroid (and retina?) are thinned, and in three places the sclera shines through between the two colobomata. Below these the fundus is stippled with pigment, but elsewhere the fundus is healthy. The Y S is lost in the coloboma. The drawing was only obtained after a most wearisome number of sittings, but the result is perfectly accurate (Fig. 4, Plate II.). V. F.: the child being so young it was found impossible to take this satisfactorily.

CASE 4 (Fig. 5, Plate III.).—Cathleen W——, aged sixteen, of London. I am indebted to Prof. Tweedy and to Dr. T. Phillips, F.R.C.S., Eng., for allowing me to make use of this case. Family healthy; six brothers and sisters, all healthy and good sight. Patient always had good health; eyes normal in appearance, pupils active. Patient came to Moorfields complaining of her eyes aching when reading. L E, V $= \frac{6}{6}$, with + 2.25 D $= \frac{6}{6}$ and J 1 at 16 inches; ophthalmoscope: fundus and disc perfectly normal. R E, V = fingers only; eye defective ever since she could remember. Refraction shows + 2 D; ophthalmoscope: disc normal, pink; vessels normal. Over region of Y S is a round-oval choroidal gap, the same size as the disc, bordered by a thin zone of pigment. The lower two thirds is nearly dense white, apparently of thickened sclerotic tissue. Two small branches of A. T. inf. pass into it and disappear, and a branch of A. T. sup. crosses it. The upper third is stippled with fine pigment. This coloboma connects the ends of a remarkable C-shaped, glistening, white patch. The upper limb appears to be entirely divested of choroid, and its inner part is limited by the sharp edge of the retino-choroid of the fundus. As we pass round the curve of the C-shaped patch this white deposit lies nearly on the surface and becomes irregularly scattered in a flocculent manner, and no free edge of either retina or choroid is visible anywhere. At the head end of the "C" patch are two nævoid-like patches of choroidal (?) vessels covered over with pigment. This pigment is quite superficial and almost obscures the head end of the "C" patch, becoming less and less in quantity towards the tail end, the last third of which is quite destitute of it. This pigment differs from the usual pigment I have observed in these colobomata, resembling that seen in retinitis pigmentosa. The choroid of the fundus enclosed by the "C" is thinned away in places, showing nearly bare sclerotic. The greater part of the C-shaped patch gives the impression of a deposit resembling that seen around the Y. S. in albuminuric retinitis. Between the coloboma and the disc, the field is traversed by innumerable radiating semi-opaque nerve fibres, while scattered here and there are a number of yellowish-white dots quite superficial. The presence of these nerve fibres gives support to the supposition that the C-shaped patch is partly congenital, but what proportion is due to persistency of medullary substance or embryonic tissue and what to disease it was impossible to determine.

The urine was tested and found free from albumen, and, moreover, the patient was in the best of health.

Case 5 (Fig. 6, Pl. III.).—Eleanor E., aged twenty-three. Patient came to Moorfield's on account of defective sight in the right eye and was kindly handed over to me by Mr. Lawson. Has never noticed any thing wrong with the left eye. Cannot be sure that she has ever seen well with her right eye, but noticed its defect by accidentally covering her left eye a few weeks ago.

V L = $\frac{6}{5}$ and J 1. Fundus perfectly healthy.

V R = P. L. only.

Ophthalmosc.: R E, disc normal. Vessels slightly tortuous, especially the veins. Arising from near the centre of the disc is a short, thick, bladder-like vessel ending in a cul-de-sac, about ½ P D long, as it appears foreshortened by the ope. Its direction is forwards and downwards, and, illuminated by the mirror, casts a distinct shadow on the fundus. It is only attached to the disc, its anterior end being quite free and movable in the vitreous. At its attachment to the disc there is distinct pulsation, and the whole vessel moves with each beat of the artery. I think I can trace its junction with the main trunk of the inf. retinal A^{y}.

If it is a persistent hyaloid artery (and I cannot account for it otherwise), it will give force to the claim of my placing the peculiar patch about to be described among my collection of colobomata.

One P D to the outer side of the disc is an oval-shaped depression, occupying the greater part of which is a tongue-shaped, nævoid-like growth, looking like an oyster half removed from its shell. The patch, 2 P D's × 1½ P D's, has its long axis vertical. The inner and outer margins overhanging the patch are white and glistening. The margins are not white from atrophic changes or deposit, as on careful focussing a great number of fine nerve-fibres are traced across them which can, moreover, be traced over the fundus for a little way. The floor of the patch around the black mass is slightly behind the edge of the patch about 1 D to 1½ D's only. It is white and glistening, the greater part covered over with fine gray pigment. Slight parallax between the outer edge and the white floor is noticed. Directed obliquely, downwards and outwards, is a nævoid-like reddish mass, covered with dense black pigment. In the centre of this patch two spots of white (sclerotic ?) shine through where the pigment mesh is open. A few fine arterial twigs cross the coloboma, but they do not curl over the edge, but pass uninterruptedly across. Below the patch and separated 1 P D from it is a crescentic flocculent deposit, quite superficial. Two P D's below this again, abutting the inferior temporal artery, is a single black spot, ½ or ⅓ P D in breadth. This spot is quite different from the "craters" so commonly seen in specific choroiditis disseminata, having its pigment almost entirely confined to the centre. (The drawing is here incorrect.) Lastly, this is the only spot to be seen in either fundus, while no history or sign of syphilis can be traced in the family. A small hemorrhage is to be seen 1 P D below and internal to the coloboma.

(Since writing this I have again examined the eye, and find now, Oct. 14, '89, that the superficial white deposit has almost entirely disappeared, while the artery which runs along the upper border of the black spot is obscured for about 2 P D's and then becomes well defined. Lastly, five small recent retinal hemorrhages can be traced to a minute artery at the extreme outer edge of the nævoid-like growth.) It may be possible that the changes seen in this case and the C-shaped crescent in the one previously described (Figs. 5 and 6, Pl. III.) are secondary to the true physiological colobomata, which, being spots of least resistance, have attracted, as it were, the pathological changes to their neighborhood.

CASE 6 (Fig. 7, Plate IV.).—Sarah S——, aged twenty-two. Patient came to me at the Western General Dispensary owing to her eyes becoming inflamed after much reading. Has never been ill in her life. Mother in good health during each confinement.

V R = $\frac{6}{6}$, with + 2 D = $\frac{6}{5}$.
V L = $\frac{6}{9}$, with + 2 D = $\frac{6}{5}$.

Ophthalmoscope: R E, disc and vessels normal. Fundus everywhere (excluding coloboma) normal. Y. S. dark red, ⅙ P D; 3½ P D external to disc, and with upper border on a level with macula, is an egg-shaped coloboma 2 P D's × 1½ P D's, long axis horizontal. The coloboma is bounded by a ring of dense black pigment, less in the upper part than the lower, where it spreads for some distance beyond the coloboma over the field in the form of a large-meshed network, in the meshes of which the choroid is thinned or nearly absent, showing white patches of clean sclerotic shining through. Between the coloboma and the Y S the field is occupied by a large group of radiating opaque nerve fibres which if traced back would meet in a focus about the centre of the coloboma. The Y S which is quite healthy appears as a cherry-red spot at the lower edge of the bundle of opaque nerve fibres just described, and half a P D internal to the coloboma. The choroid fills up the gap of the coloboma, giving it a reddish-yellow appearance, thinning to white below. It is placed, tested by refraction, 2 D's behind the retinal pigment, covering the free edge of the retino-choroid surrounding the gap. Three large choroidal vessels run horizontally across the gap, between which are minute choroidal capillaries. The choroidal vessels which run across the gap disappear from view at either end. A solitary retinal artery passes over the lower edge of the coloboma, and continues its course along the field. The pigment which covers the lower free edge of the gap is very dense and broad, and, collecting into trabeculæ, encloses a number of small gaps or spaces, which closely resemble the main coloboma except that they are much smaller in size. The upper lip of the big gap consists of a double contour or edge ; the innermost and most projecting part consisting apparently of choroid and posterior layers of the retina, while the part anterior to and just outside the deeper lip seems to consist of retina and retinal pigment only. These lips are on a different level. I think this double lip extends right round, but along its lower half and sides the dense black pigment obscures it.

The space between the two lips is quite white.

V. F. contracted, with marked marginal gaps. Well defined scotoma to electric light. For white and colors somewhat larger field and scotoma. See chart No. 3.

Case 7. (Fig. 8, Plate IV.) Same patient as the last, left eye. Ophthalmoscope: fundus almost identical with that of right eye and (excluding coloboma) everywhere normal. Y S well-defined, reddish spot with bright yellowish glistening centre ; ½ P D below Y S and just external to it is a large egg-shaped coloboma, identical in position and shape with the one in the right eye. It is surrounded by a fringe of dense pigment, which is thin below, but spreads out from its inner margin into an irregular mass occupying nearly the area of the coloboma and extending upwards to nearly the level of the Y S. This mass contains four oval gaps nearly destitute of choroid, which allows the white glistening sclera to shine through. The lower and the inner of these gaps are almost hidden by dense pigments, but the other two show the white sclerotic base about 2½ D's behind the sharp edge of the retinal pigment. The base of the large coloboma is due to an immense number of fine stippled dots, uniform in size, but which get fainter and fewer towards the centre of the patch. The lower third is crossed horizontally by several horizontal choroidal vessels fringed with thin gray pigment dots. L E, V. F. perfect in extent. Well-marked scotoma to nasal side. See Chart No. 4.

CASE 8.—Ralph T——, aged, 19 clerk. Patient came to Moorfields, (to Mr. Lawson's clinic) complaining of inability to see objects at a distance. Strong, healthy, never been ill. Teeth well developed, regular, no absence of enamel. Eldest of five children, all well and good-sighted. No history of mother having been ill before or during pregnancy.

V R with — 5 D S = $\frac{6}{9}$ and $\frac{6}{6}$ nearly.

V L with — 6 D S and — .75 D — C = $\frac{6}{9}$ well and $\frac{6}{6}$ a few letters.

Irides bluish-gray, normal. Pupils active. Both eyes well developed. Ophthalmoscope : R E, fundus shows slight thinning of choroid, and retinal hexagonal pigment round the margin of disc, especially on outer side, where there is a pronounced crescent, otherwise the fundus is quite healthy. No trace of coloboma or other defect anywhere. Y S dark red, slightly granular, with yellowish-white centre spot.

Left eye (Fig. 9, Plate V.).—Ophthalmoscope : disc somewhat smaller than in right eye, and ill defined at margin. Wedge-shaped outer triangle of disc pale bluish-white, bordered by well-marked myopic crescent, with double border edged with pigment. Fundus thinned for a considerable distance all round disc, almost reaching the Y S on the outer side. Y S situated farther away from the disc than usual—viz., 3½ P D's to its outer side. When sharply focussed a number of fine radiating striæ can be seen passing from the fovea in all directions, stippled with minute pigment dots. Commencing from the outer edge of the macula, extending horizontally outwards, is a well-defined gap 2 P D's × 1½, long axis horizontal. It forms a gap in the fundus like a button-hole stretched open ; the inner angle of which "fades" almost imperceptibly into the Y S, the outer angle being more sharply defined and covered with a black patch of pigment. The curved borders above and below are fringed with black pigment, and sharply defined, and overhanging the floor of the gap. Running horizontally across the floor are three broad anastomosing choroidal veins, which are again crossed at right angles by three fine retinal vessels which can be traced to the A. T. inf. A fine gray pigment mottling can be traced for some distance all round the patch. The whole base of the patch is pale ; the sclera shining through as a faint pink, becoming in places almost pure white, having the appearance of the fundus of an albino.

V F : No scotoma could be made out, although every possible care was taken, and the field tested separately for white, red, blue, yellow, and green. Outer half of field for white nearly normal, inner half contracted to 15°. Field for colors all equal and contracted to 60° out and 20° in. See Chart No. 7.

Case 9 (Fig. 10, Plate V.).—Arthur Y——, warehouseman, aged twenty-five, came to hospital complaining of floating threads (*muscæ volitantes ?*) when looking at the light. Four brothers and two sisters all good sight and healthy. Mother died a few years ago of Bright's disease. Patient always had good health and sight. Teeth fairly well developed, no spaces, or notches. V R = $\frac{6}{5}$ perfectly. V L = $\frac{6}{5}$ perfectly. No manifest hyperopia. Eyes well developed. Irides brown, perfect, act well to light.

Ophthalmoscope : L E, fundus and disc absolutely normal.

Ophthalmoscope : R E, disc round, very pink vessels, tortuous and glistering (shot-silk sheen), otherwise healthy. Y S healthy, surrounded by a pale halo. Fovea yellowish, pin's-point in size. Fundus healthy everywhere except below the disc, where the choroidal vessels show through the retina, in the meshes of which vessels is some very finely stippled *choroidal* pigment. In the centre of this patch of choroidal vessels, 2 P D's below and 1 P D to the inner side of the disc, is an irregular pear-shaped choroidal gap 1 P D vert. × $\frac{2}{3}$ P D horizontal. This gap is surrounded by a sharp margin, over which is distributed a moss-like layer of black pigment ending round the gap. The latter may be divided into two equal parts—an upper and a lower. Upper half shows a white sclerotic base covered with retinal pigment. The lower part of the gap is entirely filled with a mass of choroidal vessels in all respects resembling a nævus. Choroidal veins may be traced to it from all sides round the coloboma.

The retina ends sharply at the margin of the gap, although its pigment may be traced as a thin veil over both upper and lower parts of the coloboma, and is heaped up around it. The thinned choroid extends some little way beyond the scotoma. The rest of the field is quite normal.

L E, V F : Perfectly normal for white and colors.

R E, V F : Irregularly contracted above and to the outside. In the upper and outer segment is a well-marked scotoma for white. The fields for red and green are identical. They are contracted with a well-marked gap where the scotoma should be, but the latter being outside the range for colors, it was impossible to map it out. See Chart No. 5.

CASE 10 (Figs. 11 and 12, Pl. VI.).—Flora J——, aged twelve. A private patient kindly sent to me by Dr. de Carey. Patient has always enjoyed good health. Brothers and sisters, eight ; all well. No history of syphilis in the family. L E well developed, iris and fundus normal. R E about two thirds the size of L E. Iris complete, contracts imperfectly to light, enlarges only to 4 *mm* under atropine ; eyelids small, but otherwise normal. Plica semilunaris undeveloped. Slight alternating convergence. V L = $\frac{20}{30}$, V R counts fingers at two feet ; slightly improved with — 10 D. Ophthalmoscope : fundus seen best with — 9 D. Chloroidal vessels can be seen everywhere through the retina. Y. S. not obvious. No halo or fovea visible. Disc injected, ill-defined outline quite invisible below. Is entirely surrounded by a large oval coloboma with flattened ends, surrounded by a border of dense black pigment, which encroaches on the fundus below and at the inner side, but overlaps the coloboma along the outer side. Coloboma measures 3½ P D's × 2½ P D's, long axis vertical, base 2.5 D below surrounding fundus, and chalky white, showing bare sclerotic, except inside lower and outer margins, which are covered with a network of choroidal (?) vessels stippled with pigment granules. The retinal vessels all vanish as they approach the centre of the disc. They are numerous and all spring from various parts of the disc, quite independently of one another, a characteristic feature which is quite peculiar to the papillary form of coloboma, and only after crossing the coloboma do they branch dichotomously in the normal manner. Some very fine pigment-granules may be seen stippled over the field between the Y. S. and the coloboma.

V. F : Vision was too imperfect to get any field.

Case 11 (Fig. 13, Pl. VII.).—William S——, aged fourteen; errand boy. Patient healthy all his life. Teeth quite perfect, regular and even. Eyes normal size. Irides well developed, hazel-brown, pupils contract well to light. Slight nystagmus L E. No history or signs of syphilis in the family.

V R with − 1 D S and + 1 D C axis vertical = $\frac{20}{50}$.
V L with − 1 D S and + 1 D C axis vertical = $\frac{20}{50}$.

R E. Ophthalmoscope: disc round, rosy-pink, vessels normal, well developed. At the outer part of the disc, between the two temporal arteries (A. T^s. and A. T_i.), where they emerge from the centre of the papilla, the latter appears deficient, and in its place we see none of the rosy-pink of the disc, but a dense white base covered with four or five orange-red vessels, which are seen to be continuous choroidal vessels immediately external to the disc, which are plainly seen for some distance (1½ P D's), owing to the thinning of the overlying retinal pigment layer.

At 2 P D's external to the disc is the inner boundary of an immense coloboma measuring 5 P D's each way. This patch fills up nearly the whole space between the superior and inferior temporal arteries. Indeed, the whole character of the fundus is altered between these two arteries, from their commencement to as far outward as they can be traced with the ophthalmoscope. Thus at the centre of the disc we notice at least five choroidal vessels crossing the disc between the two temporal arteries. Tracing the fundus outwards the choroidal vessels are very evident, and the retinal pigment is more or less absent.

Just external to the large coloboma are two more colobomas with a trace of a third. The large coloboma is sharply defined by the edge of choroid, which forms a free edge all round the coloboma. The portion nearest the disc is glistening white and nearly circular, and is situated 3 D's behind the tissue on the margin of the patch. Here the parallax is strongly marked. The patch is bounded externally by a large nævoid mass of choroidal vessels. This structure occupies about half the patch and is covered by a loose veil of retinal pigment. This latter forms the usual ring round the free edge of the coloboma. The upper part of the coloboma is lobulated, and the pigment fills up the recesses between the lobules, and passing between them in the form of trabeculæ, joins the central network of pigment which covers the nævus. Here we may notice the same structure which is so well marked in some of the other cases (see Fig. 2, Pl. I., and Fig.

14, Pl. VIII.), viz., a central nævoid mass covered with a veil of pigment, which latter is connected with the border circle of pigment by trabeculæ; these divide the white zone outside the nævus into numerous lobules as in the cortex of a lymphatic gland. These lobules vary in size and for the most part are oval, with their long axes to the centre. A large number of choroidal vessels may be seen passing from beneath the upper free edge of the patch in a direction parallel to and behind the trabeculæ, to join the meshes which make up the nævus.

Just external to the large coloboma are three small ones. The innermost of these is the best defined. Its base is 2 or 2½ P D's below the surface and crossed by choroidal vessels, and patches of pigment connected with the usual bordering. These three small colobomata differ from most of the others I have examined, inasmuch as the choroidal vessels can be traced up to and beyond the patches, and not merely across them to disappear beneath the margin. This may be explained by the choroid being everywhere visible round about the patches, owing to the scarcity of retinal pigment. A similar coloboma to these is to be seen immediately at the foot of the large coloboma. A retinal artery crosses its outer end.

Case 12 (Fig. 14, Plate VIII.).—Same patient.

Left eye.—Disc round, rosy-pink, seen best with + 0.5 D S. Vessels normal. In the triangle formed by the two temporal arteries, as they diverge from the centre to the apparent outer margin of the disc, the disc seems to be wanting, and its place is occupied by a chalk-white background covered with several choroidal vessels, and over all three curved, rod-like patches of retinal pigment. The appearance of this part of the disc closely resembles the corresponding portion of the disc in the opposite eye. It consists of choroid and retina, and differs entirely from the rest of the disc.

Half a P D to the outer side of the disc is the commencement of a large, irregular-shaped coloboma, 4 P D's × 2¼ P D's, which is exceedingly well defined and sharply bordered. Its long axis (4 P D's) is directed obliquely upwards and outwards. Half a P D external to this patch, and in a line with its long axis, is a second patch, 3 P D's square. It gives one the idea as being part of the former patch, a bridge of normal retino-choroidal tissue separating it off. The disc is seen best with + 1.5 D, the white floor of the first-mentioned white patch with − 1 D (direct), the base of the outermost (square) patch with − 2 D.

Inner coloboma.—On the inner curved border of the coloboma two clusters of pigment may be observed passing for a short distance across half of the coloboma. These are evidently the remains of trabeculæ, which stretched across the ectasia to the pigment network covering the nævus. The layer of pigment on the inner side of the gap is separated from the tissue outside by a whitish border of thinned choroid. A few choroidal veins may be seen crossing the patch to enter the nævus ; a branch from each outer retinal artery passes across both patches.

The outer half of the coloboma is chiefly occupied by a large nævus, which is made up of four thick processes of nævoid tissue, which spring from the opposite sides of the coloboma, and unite in the centre to form a dense red mass of choroidal vessels. The limbs with the outer border enclose three egg-shaped gaps, similar to the big inner patch above described, only more covered with pigment. A dense network of retinal pigment spreads itself over the whole of this half of the patch, forming the characteristic trabeculæ which pass in festoons towards the centre network.

The outer coloboma closely resembles the inner half of the coloboma described above. It is surrounded by a well-defined, free edge of retina and choroid, but the pigment is scanty. There is an attempt at the formation of a choroidal nævus at the outer half of the patch, but it is not covered by any pigment. A few choroidal vessels appear in the upper half, while five or six branches of the retinal arteries pass onwards across the gap. *It is curious that, although the arteries pass freely across, the veins do not do so, nor have I noticed any case as yet in which a retinal vein has crossed an extra-papillary coloboma.* It is true the veins do so in Fig. 12, but that includes the disc.

The sclerotic floor of this patch is seen with — 2 D, and the edge with + 1.5 D, making 3½ D between the two levels.

V. F. : Contracted for white and colors to outer side. A large marginal gap contracts the field in the upper nasal quarter, corresponding very roughly only with the coloboma. There is no scotoma. See Chart No. 8.

I cannot conclude this article without acknowledging my indebtedness to Mr. Lawson and Mr. Tweedy for their great kindness throughout, and for the liberal way in which they have permitted me to make use of their cases.

AMETROPIC CHOROIDO-RETINITIS (" CENTRAL CHOROIDITIS ").

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THE etiology of the peculiar form of choroidal disease called central choroiditis seems, by common consent, to be a mystery. Hutchinson classifies all choroidites into twelve groups, of which the third and fourth alone concern us in the present inquiry. His third class is " Senile Central Choroiditis, or Tay's Choroiditis, always central, and met with only in those past middle age"; the fourth, or " Hereditary Choroiditis, various in form, often beginning in childhood, but sometimes not till middle life; several members of the same family are affected, and the changes are usually aggressive. Tay's choroiditis may sometimes occur as a family malady." I have selected from my records the clinical details of the following eight cases to illustrate my theory that central choroiditis is due to ametropia. As they are neither senile nor hereditary they do not fall into Hutchinson's classification.

CASE I.—N. J., a young man aged twenty-five, otherwise in excellent health, complained of growing inability to read or write, except for but a minute or two at a time, and by a strong light. He could not read a line at night or in a dark day. There was no ocular or cerebral pain, though the irritative and reflex symptoms usually accompanying eye-strain had in previous years been severe. From this and other symptoms I concluded that his present myopic refraction had but recently come on, and that in the beginning of his retinal failure he had been a hyperope. His acuteness of vision was but about $\frac{20}{200}$, and not improved by correction of the ametropia. The maculæ, especially above, were surrounded by nearly concentric strata-like clouds of dark-brown pigment patches, extending about 15° outward. Both eyes were about equally affected. The family and personal histories were excellent, hereditary disease and syphilis being almost certainly excluded. He was a civil engineer by profession, and had been a hard student. I advised him to give up his profession and take to farming or some similar occupation. He is now trying ranch-life in Colorado, and vision does not now, as formerly, seem to be deteriorating.

Case 2.—Dr. G. J., aged twenty-eight, complained only of slight asthenopic symptoms, and that the eyes soon tired with near work, especially by artificial light. A hasty ophthalmoscopic examination showed nothing more than compound hyperopic astigmatism, and this seemed to explain the symptoms and dictate the treatment. I was accordingly surprised to find that the most careful refraction brought out only a doubtful $\frac{20}{30}$ vision. A more careful ophthalmoscopic search solved the riddle, as I believe it would often do in many cases of "amblyopia." Both maculæ were clouded and surrounded by a tawny brownish stippling, differing in character entirely from the pigmentation of the rest of the fundus, and plainly pathological. The family and personal history was above reproach. His refractive error—mixed and hyperopic astigmatism of 1.00 D at unusual axes—was at once corrected and all possible avoidance of near work ordered. In addition to his professional studies, his specialty had long demanded much continuous ocular labor of an exacting nature.

Case 3.—Miss C., aged twenty-two, of healthy parents and history, complained that her eyes tired quickly with near work, and especially in the evening, and also of the usual symptoms of hyperopia. Normal vision—R $\frac{20}{40}$, L $\frac{20}{30}$. Under a mydriatic I found a simple hyperopia of 4.00 and 3.50 D respectively. With these lenses the vision with strong light was with either eye doubtful $\frac{20}{20}$, but with the least weakening of the light the acuity of the right eye quickly fell. The pathological stippling or pigmentation about the macula of this eye was so slight that unless one looked for it carefully, and knew about what to look for, it would easily be missed. The left is normal in this respect. The degenerative process was in this patient in its earliest stages, and it will be of interest to watch the future developments, whether the glasses prevent further deterioration, etc.

Case 4.—Mrs. K. B., aged nineteen, complained of headache and asthenopic symptoms, quickly made worse by near work. Normal vision, R $\frac{20}{20}$ (?), L $\frac{20}{50}$. The refraction, under a mydriatic, showed R + sph. 0.75 D, ⊃ + cyl. 0.50 D, ax. 75°; L + sph. 0.50 D, ⊃ + cyl. 0.75 D, ax. 105°. But with these lenses the acuity of the left eye is still only $\frac{20}{50}$; that of the right being $\frac{20}{20}$. While looking at the $\frac{20}{50}$ line, one letter of the line is not seen at all, whilst the letter at either side is readily detected. The pigmentary changes about the left macula were marked, the stippling had often become confluent, and formed coarse brown splotches in some places, and there was an unusual lack of symmetry or concentric character in the grouping around the macula. No such changes were recognizable in the other eye. Family and personal history showed no disease or diathesis.

Case 5.—Miss P. N., a seamstress, aged forty-three, complained of tired eyes after work. Normal vision, R $\frac{20}{20}$, L $\frac{20}{40}$. The refractive error of hyperopia, one dioptry, having been corrected, the right eye regained perfect acuity, but the left was improved only a very little. She had sewed every day for many years; had had good health all her life, and her parents had enjoyed good eyesight and health up to old age. The left macula region was typically pigmented, the right normal. There is also in the left papilla a pretty sharply delimited bundle of atrophic fibres, upon the temporal side, having a dead bluish-white appearance, and comprising perhaps one fifth of the total papilla space, and at a position corresponding to that of the gathering of the macula fibres.

Case 6.—Mrs. P. L., aged thirty-two, used formerly to be a great reader at night, and by a common oil lamp, after sewing much of the day. The eyes finally began to tire, until of late she can read but a little while at a time, except by daylight. At night the eyes tire, and the image fades after working or reading a few minutes. Vision, without a mydriatic: R $\frac{20}{50}$, L cannot count fingers; with a mydriatic; R $\frac{20}{50}$ + sph. 0.62 D, ⊃ + cyl. 0.25 D, ax. 90° = $\frac{20}{20}$; L + sph. 2.50 ⊃ + cyl. 1.00 D, ax. 90° = $\frac{10}{200}$. The ophthalmoscope reveals an advanced and extensive pathological pigmentation of the left macula region, extending in this case farther toward the equator of the eye than I had ever seen it. The stippling of the right was limited, and delicate in the extreme.

Case 7.—Wm. J., aged thirteen, has had headaches for several years, and made worse by reading. He is an ambitious, brightminded school-boy, and has studied hard both at school and at home at nights, until of late he at once gets sleepy when he tries to study by artificial light. The refraction and the ophthalmoscope explain both symptoms. With paralyzed accommodation I find both eyes alike in acuity, refraction, and a typical incipient ametropic chorido-retinitis. Refraction: $+$ sph. 1.75 D, $\supset +$ cyl. 3.00 D, ax. 90° $= \frac{20}{30}$.

Case 8.—Miss A. L., aged twenty-five, has for five years been wearing Sph. $-$ 1.75 from a prominent advertising optician of this city. She has compound hyperopic astigmatism, and has suffered much from the artificial hyperopia and retinal injury directly produced by this "scientific" optician. The proper correction of her ametropia gives her a visual acuity of $\frac{20}{50}$ and $\frac{20}{30}$. Both maculæ show the typical pigmentary degenerations—the right the more pronounced.

The emphatic teachings of these and other cases that have come under my care are: (1) the absence of all systemic, toxic, or hereditary conditions that might be or that are commonly adduced in a vague sort of way to account for the ocular lesion; (2) the existence of an irritating ametropia that threw a blurred or abnormal image upon the macula; (3) the exaggeration of the eye-strain by long-continued and severe ocular labor at near-range.

As to senile central choroiditis, since I first recognized the connection of ametropia and the choroido-retinal lesion, I have had but few cases of central choroiditis in the old, but in these there also coexisted a considerable refractive error, and of course with presbyopia—also uncorrected. It is plain that presbyopia, if uncorrected or improperly corrected, even without other refractive error, would induce the condition that seems to me to play the chief etiological rôle—*i. e.*, an habitually imperfectly formed retinal image. Therefore, central choroiditis may be senile, the result of presbyopia, and still be due to precisely the same essential cause as in adults and the young.

Concerning the existence of the lesion in the young, I have never seen a case of central choroiditis in children that showed any thing but the slightest stippling. I have no doubt I have passed by many such however. Our school systems must produce them more frequently than supposed, but in such cases the difficulty of detecting such a lesion is greatly increased. At best the defect is difficult to examine, is overlooked because unsuspected, and is so easily classed as amblyopia. In cities where schools and high-pressure force the young and ametropic eye to an excess of work, early visual troubles bring the child into the oculist's hands, and his prescription stops the growth of the macular disease. That chronic eye-strain *produces* lessened acuity is a truism of ophthalmic practice ; an undiscovered pigment-change at the macula would and does *explain* it. But in this connection I cannot forbear availing myself of another's work, and though he is an unconscious and perhaps unwilling witness, his conclusions seem powerful testimonials in behalf of the theory above suggested. I allude to the article by Edgar A. Browne on " Central Choroiditis, with Slight Cerebral Symptoms in Children," in the *Royal London Ophthalmic Hospital Reports*, January, 1889. The argument there advanced is that these cases are connected with and due to slight attacks of meningitis or other cerebral disease, " just as similar symptoms are allied with optic neuritis." This seems a wholly gratuitous, if not forced, assumption, and a non-convincing analogy. Since Deutschman's investigations, we can understand how coarse cerebral disease may induce papillitis, but one can only wonder how a slight and passing meningeal hyperæmia could produce intimate and localized choroido-retinal degeneration. The symptoms of these cases, as given by Mr. Browne, are night-terrors, gastric derangements (vomiting, loss of appetite, etc.), constipation, choreic movements, etc. But surely every ophthalmic surgeon knows these are the commonest symptoms of simple uncomplicated eye-strain in the young. Apply proper spectacles to such patients, and the night-terrors, nausea, nervousness, anorexia, and headache disappear as if by magic. This is not hobby-riding, or mistaken enthusiasm, or *post hoc—propter hoc* logic, but undeniable fact. According to my theory and belief, the " slight attacks of meningitis," are the common reflex-neuroses of eye-strain, and the choroido-retinal lesion the result of an abnormal stimulus,—both sets of facts the consequence of a single cause—ametropia.

Without any violence to the facts, I think the existence of this peculiar choroido-retinal lesion, whether in the young, the middle-aged, or the old, whether hereditary or not, may be correlated and ranged under the one etiological factor. The existence of any considerable number of cases, that have arisen without ametropia, would invalidate the theory. I have never seen a single instance. Neither have I, since the theory took shape in my own mind, ever seen a case of long-standing uncorrected hyperopia or astigmatism too great for the ciliary muscle to neutralize, that did not show the pathological pigmentation and the subnormal acuity,—subnormal at least by weak light in the early stages, little or not at all improvable by lenses, in the later stages. It may be said that entirely too few illustrations exist as a supposed result from a widely acting cause. Ametropia is immensely common, central choroiditis very rare. To this it may be replied that we have no idea how common the early stages of central choroidal disease may be. It may be doubted if oculists examine the macular region of all their patients with great accuracy, and the blanket-name for a mystery, amblyopia, doubtless covers a multitude of negligences. The location of the lesion also points to some causal relation with the act of habitual vision. It must also not be forgotten that the strain of civilization upon the naturally hyperopic eye is not yet on to the full. It remains to be seen whether that eye will remain so resistant in the future as in the short past in which it has been tested. Moreover, we must remember that change of occupation constantly sifts out those eyes that cannot withstand the strain of near work, and sets them easier tasks; that the growing practice of applying spectacles to the young checks the retinal deterioration; and lastly, that the varying resisting power of retinæ, like that of other tissues, produces exceptional and apparently unaccountable immunity in withstanding the attack. It is to be supposed that the brunt of such an attack is always borne by the ciliary muscle. The Müller ring-fibres are the response to the excessive stimulus. With continuous near work and uncorrected ametropia this resource fails, and the retina is then subjected to an abnormal and pathogenic stimulus. Central choroiditis, with its resultant atrophy of the nerve-fibres and subnormal visual acuity, is the reaction and result of this unaccustomed and unnatural stimulation. It is but one illustration of the universal physiological law, that functional reaction to new stimuli develops modification of structure, and if the new stimulus is so abnormal that physiological reaction and adaptation cannot take place to resist and preserve, then the organ must suffer, and even atrophy. Were it not for the beneficent work of spectacles, it might reasonably be doubted if civilization would not, in part at least, bring itself to an ending in the results of severe eye-strain ; that is, in myopia, ocular and nervous disease, headache, chorea, etc., etc., and, more important still, in the mal-assimilation that results from reflex derangements of the digestive system, primarily caused by eye-strain.[1]

In order more clearly to bring out the characteristic features of my conception, let me briefly epitomize the different aspects of the disease :

NOMENCLATURE. — Though the lesion, like so many others covered with the indicative suffix—*itis*, is evidently extremely chronic, and in no true sense inflammatory, it is plain that the habitual terminal, *itis*, must be allowed. Again, every thing indicates that the lesion is retinal, and not choroidal. At the best we may qualify it as a choroido-retinitis. Since it is known that the pigmentary layer, both functionally and evolutionally, belongs to the retina, and not to the choroid, the error of calling these and certain other fundus changes, choroidal, becomes all the more apparent. But, under protest, the habit must be allowed to obtain. According to the theory, these macula-changes may be due to any one, or to any combination, of four factors—uncorrected hyperopia, astigmatism, presbyopia, and insufficiency. Either of these conditions, not corrected by the excessive action and neutralizing power of the ciliary muscle, nor by spectacles, supplies the retina wlth an abnormal stimulus. We cannot say the choroido-retinitis is due to eye-strain, since so long as the eye-strain is sufficient the dioptric mechanism supplies a proper image. It is when the task is too great or too continuous, and the ciliary muscle strikes work, throwing the labor upon the retina and the cerebral mechanism, that the retinal degeneration begins. Hence, the suggestion that the usual reflex consequences of pure eye-strain are greater prior to the retinal deterioration than after it has begun. The dulling of retinal sensibility also reacts to the same end, by not demanding of the ciliary muscle a clear-cut image. It is only by a circumlocution that we can express the diseased condition; we may call it a macular choroido-retinitis, due to abnormal focalization of the image or light-stimulus,—or, briefly, ametropic choroido-retinitis.

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[1] See, for example, my article in the January, 1890 number of the *American Journal of Medical Sciences*.

DEFINITION.—Chronic changes in the macula region, chiefly in the retina, but possibly implicating the choroid, occurring in the young or old, but generally in young adults, caused by long-continued ametropia (abnormalism of the image or light-stimulus), evidenced objectively by pigmentary changes, and subjectively by subnormal visual acuity, retinal asthenopia, and lessened reaction to weak light.

ETIOLOGY.—These changes are primarily produced by a long-continued, non-physiological stimulus, that slowly produces a functional abnormalism in the molecular and organic reactions of the retina, and that finally ends in organic tissue-change. The location and limitation of the lesion points to a connection with the exercise of habitual vision, and its association with chronic uncorrected ametropia affords clinical proof of the assumption. It is only natural that structures so highly and complexly differentiated and of such amazing delicacy of adjustment should be influenced pathologically by an abnormal stimulus. It has long been a source of wonder that the retina was little subject to idiopathic degenerative and inflammatory changes. I question if this supposed immunity is so great, or if it will remain so great. I believe this lesion is far more frequent than supposed, and that it will become yet more so. The habitual and evolutional function of the retina is its peculiar response to a marvellously delicate and finely adjusted, properly focussed, image. Its task is to transform light-waves into nerve-force, the change of etherial into neural vibrations. In this work the retinal pigment cells are a *sine qua non.* Their existence precedes even the most primitive type of eye in the lowest animal. How they act is a mystery, but without them vision is impossible. It could hardly be otherwise than that a severe, long-continued, and abnormal stimulus, to which, prior to the invention of printing, the retina was never accustomed, and that is now acting with almost malignant exaggeration, should finally produce some such result as this macular choroido-retinitis.

PATHOLOGY.—Histological examination of the changed tissues is greatly to be desired. So far as I know no such studies by accurate and competent hands have been made. The fact that we know nothing of the intimate nature of the physiological retinal function precludes exact knowledge of pathological modifications. It may, indeed, transpire that the abnormal changes of the retina are secondary to changes in the cortical or trophic optical centres. The nerve messages from the macula must necessarily be inaccurate and functionally imperfect, and the labor of transforming them into normal sensations by the central cerebral cells must be greater, and may be reasonably supposed to produce abnormalism there, which might react upon the retinal end-organs and induce secondary functional and organic changes in them. Whether this be so or not, and whether such changes are trophic, inhibitive, or failures to adjust, etc., must for the present remain undecided. They are certainly morbid, and their objective manifestation consists in pigmentary changes; the subjective, in loss of visual acuity and power. These things, therefore appear to me indisputable: The retinal function is necessarily bound up with that of the pigment cells; long-continued ametropia, not neutralized by spectacles or by the ciliary muscle, produces pathological changes in the pigmentation of the macular region, and a loss of visual acuity and power in proportion to the extent of the pigmentary abnormalism. The physiological production of light or color out of ether-waves requires the intermediation of peculiar colored organs in the retina. A chronically abnormal condition of the ether waves or chromatogenic stimulus would almost necessarily produce pathological conditions of the colored organs. Abnormalism of stimulus everywhere results in pathological reaction.

OPHTHALMOSCOPIC PICTURE.—I have found that the peculiar brownish stippling of the macular region may precede any great loss of visual acuity if the eye has its refractive error corrected and the object be illuminated with a strong light. At this time the pathological change may be easily overlooked. It is difficult to observe the macula when the person is young and the visual acuity perfect. The differential diagnosis between physiological and pathological pigmentation of the macula, is the most difficult task in ophthalmoscopy, and requires the most exquisite judgment and delicacy both of physical and mental technique. When at a later stage the tiny dots of brownish discoloration have become confluent and more pronounced, their discovery becomes more easy. Examined attentively and by certain lights and lenses of the ophthalmoscope there may be seen bright or dull white dots or sinuous irregular white streakings, very small and delicate, interspersed among the brownish stippling and layers. Rarely these seem more noticeable than the pigment anomaly. In almost all cases the pigmentary changes show some sort of symmetrical or concentric relations with the macula, and do not usually extend beyond a radius of ten or twenty degrees. I think I have noticed that the more pronounced changes are above and to the outer side of the macula,—corresponding to the position of the most continuous stimulation by the retinal image. The fovea itself shows more decided pigmentation. In advanced stages the circumjacent pigmentation assumes a stratified appearance like clouds about the setting sun, only the color, a tawny dark brown, is darker than in earlier stages, but is never black like that in other types of true choroidal inflammations. With an increase of the morbid changes there is a synchronous atrophy of optic-nerve fibres supplying this region. The remainder of the fibres and the peripheral field of vision remain normal in extent and function.

Symptomatology.—The usual reflex symptoms of eye-strain will be found inversely proportional to the amount of permanent loss of visual acuity, or what amounts to the same thing to the amount of retinal damage. The greater the amblyopia the less the headache, gastric disturbance, etc. The chief and constant complaint is of inability to continue near work except for short periods (which is exactly in ratio of the macular injury and pigmentation), and by means of a strong light. The image cannot be held, fades out, the eyes tire, lachrymation and other symptoms of irritation follow if the suffering retina is forced to continue its work. The loss of acuity is not generally remediable by lenses, though the correction of ametropia is possible, the patient clearly distinguishing between better or worse, while not being able to decipher a smaller letter. The loss of acuity is exactly in proportion to the amount of pigmentation about the macula as shown by the ophthalmoscope. Both eyes may be affected, and in different degrees, or but one alone, and the eye that has had the most imperfect image is the one usually showing subjectively and objectively the greatest abnormalism.

Prognosis.—Though my experience is too limited to be dogmatic, I believe that at most any stage of the disease the visual acuity then remaining may be permanently retained upon the condition that the treatment be rigidly followed. I doubt if any recovery of the lost visual power is possible.

Treatment.—The treatment is simple, and consists in the most perfect correction of the ametropia possible. Eye-strain must be permanently relieved. In addition to this, in cases when the acuity, we will say, has fallen as low as $\frac{20}{40}$, there should be permanent renunciation of all near work, at least except by aid of a strong white, steady light, —that is, by clear daylight. Whatever near work be allowed must also be done by bits, as it were,—a short period of near work followed by a long period of rest.

SUMMARY.—Chronic uncorrected ametropia may result in permanent lesion of the macula region, usually accompanied by pigmentary changes, with deterioration, probably permanent, of visual power and acuity. "Central choroiditis" is explainable in this way. Simple myopia is the least apt to produce the lesion, and myopic astigmatism, simple and compound, comes next in order. Simple hyperopic astigmatism is next in the hierarchy, followed by simple hyperopia, whilst compound hyperopic astigmatism, especially if complicated with insufficiency, occupies the highest place as an etiological factor.

Apart therefore from the grave systemic and cerebral reflex disorders (dyspepsia, mal-assimilation, headache, chorea, etc., etc.) produced by "eye-strain," there is great risk to the ocular mechanism itself—to its most vital and essential part—in leaving ametropia chronically uncorrected.

A NEW DRESSING FOR CATARACT-EXTRACTION AND OTHER WOUNDS OF THE EYEBALL.[1]

H. GIFFORD, M.D., Omaha, Neb.

(*With two wood-cuts.*)

THE main desiderata in a cataract-dressing are three: (1) Support to the lips of the wound, which runs no risk of distorting the natural curves of the eyeball. (2) Protection from accidental violence. (3) As complete rest to the eye as possible. For the purpose of the present discussion, the methods of dressing now in vogue can be considered to be two: the ordinary method, in which a roller or other bandage holds absorbent cotton against the outer surface of the lids; and the open method of Michel[2] and Chisolm, in which no bandage is applied, the lids simply being held closed by a strip of isinglass plaster. Neither of these methods accomplishes the objects above mentioned with any thing like theoretical perfection. The open method has an undoubted advantage over the bandage, in putting no pressure upon the globe beyond that exercised by the eyelids, the most perfect splints possible. No matter with what care we may distribute it, cotton cannot be placed next the lids and a secure bandage applied without more risk of distorting the eyeball than the unaided pressure of the lids would present. Experience shows that this risk is very slight, but occasional cases, where the anterior chamber is not promptly re-established, and the occurrence of infections of the wound and anterior chamber (which must be favored by any lack of coaptation of the wound-lips), show that it is by no means imaginary. The risk, however, is one which the majority of surgeons choose, in preference to that of leaving the eye so uncovered that a slight accidental touch on the lids might reopen the wound, to say nothing of more serious damage which might result from greater violence. This risk is also slight, as the results of the advocates of the open treatment prove, but it is even more demonstrably real than that of uneven pressure from the ordinary bandage. The dressing which I propose allows the advantage of the open treatment, at the same time affording far more security against accidental violence than the ordinary bandage. On completing the operation, the lids are closed with a bit of plaster, as in the open treatment; then a concave shield of stiff paste-board is applied, of such size that the flaring edges rest on the bridge of the nose, the forehead, the temple, and the cheek, while the arched centre remains an inch or more from the surface of the lids. This is held firmly in position, partly by an elastic reaching behind the head, and in addition by a firm

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[1] Read in part at the Missouri Valley Medical Society, September 20, 1889.

[2] Dr. Hobby, of Iowa City, informs me that before the communications of Chisholm and of Michel, he had begun using the plaster dressing, following the practice of Haynes Walton, of the last century; but that he gave it up on account of danger from the lack of protection.

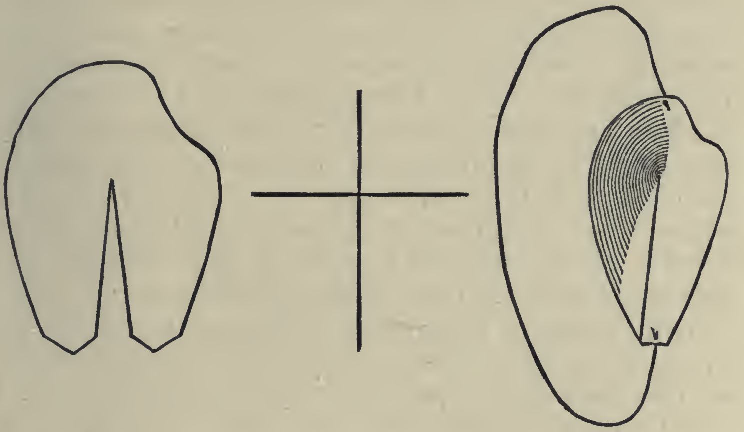

FIG. 1. FIG. 2.

Fig. 1: Shape of paste-board pattern. Fig. 2: Shape of same after having been wet and bent into shape for the right eye.

monocular roller bandage. The other eye is then closed with a thick wad of cotton, kept in place by a pasteboard shield, held on by an elastic alone. This dressing, it is plain, while involving no risk of unequal pressure on the globe, affords much greater protection than the ordinary bandage; for the latter might easily allow to be transmitted to the eyeball the pressure of a bump or blow, which would make no impression at all on the stiff arch of the pasteboard shield. The unoperated eye is bandaged, because by this alone can the most perfect rest be obtained for the other eye; but it is bandaged separately, in the simple way described, because in any given case an emergency may arise, rendering the sudden removal of the obstruction to such sight as the unoperated eye may possess, a real or apparent necessity; real, as in the case of an outbreak of fire in the house; imaginary, in the fright or delirium which sometimes overtakes old people when deprived of all light. In such cases the patient would naturally tear off the easily removed shield from the unoperated eye, and thus at once regain such sight as was possessed before the operation, without, in any way, disturbing the dressing of the operated eye.[1] The separate bandage for the unoperated eye is also to be preferred on account of its permitting the eye to be washed without disturbing the other eye, or even raising the head from the pillow, a matter often of considerable importance to the comfort of the patient, the itching of the unoperated eye sometimes being the main cause of complaint.

With regard to the method of keeping the lids on the operated side closed under the shield, the surgeon can suit his own prejudices. The simple plaster may be used, or a bit of parchment paper or lint covered on one side with an antiseptic ointment may be placed along the palpebral fissure and the plaster fastened above and below this; or, if he prefer a moist dressing, he can use a tuft of cotton soaked in a sublimate solution, so loose as to conform of its own weight to the surface of the lids, a bit of dentist's rubber being laid over the cotton, large enough to reach out beyond the edges of the shield.

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[1] It is perhaps an open question whether the greater chance of delirium when both eyes are closed, with consequent greater danger even with a separate, easily removed bandage for the second eye, does not more than counterbalance the disadvantage of leaving this eye open.

If the advocates of the open method insist, as some seem inclined to do, that a comparatively free access of light to the eye contributes to the freedom from photophobia, which their cases exhibit, this furnishes no excuse for rejecting the protection afforded by the shield, as it is very simple to cut a hole in the latter for the admission of light, arranging the folds of the bandage so as to leave the hole uncovered. On the other hand, if any persist in preferring what I consider the objectionable practice of binding a wad of cotton more or less tightly against the lids, the shield will distribute this pressure more evenly than the bandage alone, beside giving much better protection. Of course, any stiff substance can be used for the shields. Permanent ones[1] of hard rubber or metal can be made; but for practical purposes, pasteboard answers as well, or better. A nurse can make a dozen in an hour after the pattern figured above.

The application of the shields does not by any means end with cataract cases. They can be used with advantage in all cases of extensive wounds of the external eye tunics. With young children, where bandages are so difficult to keep in place, the shield allows the bandage to be applied tightly enough to hold it perfectly secure, without the slightest danger of undue pressure on the globe. In monocular gonorrhœa, also, the shield can be used to protect the non-infected eye, a fringe of cotton being fastened, with collodion to its edges and to the skin, and a hole being cut in the centre for visual purposes.

To sum up, the following propositions seem, to me, undebatable: (1) The coaptation of a cataract-extraction wound is less apt to be disturbed by the pressure of the lids alone than by that of cotton pressed against the lids by a bandage. (2) The possible danger from accidental violence cannot be gainsaid, and the shield-dressing affords a surer protection against such violence than any other method at present used. (3) The closure of both eyes is necessary to secure the most perfect quiet to the operated one. (4) There is some advantage in being able to remove the covering of the unoperated eye without disturbing that of the other. The logical consequence of admitting these propositions is the conclusion that some such dressing as that above described is better than those now in vogue. The theoretical advantages of the shield-dressing are so plain, and the difficulty of establishing them with any certainty, by the statistical method, would be so great, that the writer has ventured to make this communication after having used it in a few cases only.

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[1] Similar shields have been proposed by Wicherkiewicz, very rationally, it seems to me, as a substitute for the dark-room cure.

A REMARKABLE CASE OF DETACHMENT OF THE RETINA—RECOVERY.

By J. H. THOMPSON, M.D., Kansas City, Mo.,

PROF. OTOLOGY KANSAS CITY MED. COLLEGE, LATE PROF. CLINICAL OPHTHALMOLOGY AND OPHTHALMIC SURGEON TO THE CITY HOSPITAL, SECRETARY OF THE OPHTHALMIC SECTION AMERICAN MED. ASSOCIATION, 1887.

IT has been my good fortune to have seen in the last eighteen months several very interesting cases of diseases of the eye, one of which was so extraordinary in many respects that I believe it sufficiently important to record.

Mr. H. Smith, aged twenty-five, an electrician, was wounded while hunting, April 29, 1888. He and a companion were in a large open swamp, shooting snipe, when his friend accidentally discharged his gun, and he received the charge of shot in the face. They were distant from each other at the time about thirty yards. He immediately found that he was blind, was led off the field, walked five miles to the station, then rode fifty miles in the cars, and reached home that night. I saw him early the next morning. He was then suffering from many shot-wounds in the face and head ; the skin was much swollen, especially the lids of both eyes, which were ecchymosed and covered with a bloody discharge from the orbits. By separating the lids with elevators, I found that the right eye was collapsed, filled with blood, and perforated in three places. One opening was in the centre of the cornea, another at the corneo-scleral edge, and another at the opposite meridian, which was quite large, and from which escaped a quantity of vitreous. The other eye was also badly damaged ; it was partly collapsed, the anterior chamber filled with blood, and showed a shot-wound at the outer inferior zone of the ciliary region, about three lines distant from the cornea ; the wound corresponded to an opening through the lower lid. It opened into the interior of the eye, and from it exuded a bead of vitreous humor. He had fair perception of light, and there was no sense of pain in either eye.

That morning he was admitted to the All Saints Hospital, given an anodine, and put to bed. The eyes were then covered with cloths dipped in a cold solution of the bichloride of mercury. The following morning I removed the right eye, because of a beginning panophthalmitis, and while the patient was under the anæsthetic examined more carefully the left. The only wound in the organ was the one before mentioned. Without probing or turning the eyeball over a few degrees I was uncertain of the full extent of the injury, but, because of a clot deep in the orbit near the sclera, I was of the opinion that the shot had passed entirely through the eye, hugging the inner surface of sclera in its passage from before backwards. An ophthalmoscopic examination could not be made, because of the interocular hemorrhage and the clot in the anterior chamber. Of course at the time the tension of the eye was nil.

Inasmuch as there was nothing more that could be done, the patient was confined strictly to his bed in a dark room, and the eye simply dressed with antiseptic cotton, under a light compress bandage. In one week the wound had apparently healed, the blood disappeared from behind the cornea, the tension had increased to near the normal, and the interior of the eye open to the ophthalmoscope. I then saw in the eye about the wound a mass of blood ; otherwise the vitreous was transparent, although somewhat clouded. The perception of light was good ; fingers recognized a few feet ; the visual field only abolished over an area corresponding to the size of the internal clot. The treatment and rest were continued. The case progressed favorably.

The following week the eye was again examined. The blood had materially diminished ; the vitreous was much clearer than before ; the tension was normal ; there was a little conjunctival congestion, but no symptoms of ciliary irritation ; the scotoma was smaller, and the patient able to see large print.

I had seen quite a number of shot-wounds of the eye, and had learnt from experience that such injuries are nearly always fatal to vision, but because of the favorable progress of this case I was inclined to think that perhaps the eye might recover.

The same careful management was continued, the patient kept in bed on his back, and not allowed to leave it for any purpose.

At the next examination, about one week later, the eye was apparently well ; the picture of the fundus was normal ; the disc clear and distinct ; central vision good ; could read the finest print, and see the second-hand of a watch move several feet distant ; the reaction of the pupil was natural ; no ciliary injection ; no signs of irritation anywhere within the orbit ; interocular tension normal. The patient then left his bed, but not the room ; the dark screens were removed, and a moderate quantity of light permitted to enter. The bandages were also removed, and a shade substituted.

A few days later the eye was again examined, when I saw the ominous reflex of a beginning detachment of the retina. It appeared to start at the side of the eye above the wound, and was limited to that neighborhood. The bandages were reapplied, and the patient sent to bed again. All that could be done was powerless to stay the progress of the detachment ; from hour to hour it increased, coming on from both sides of the equator, and finally closed over the disc, rendering the patient blind. Sight gradually disappeared. On June 20th the young man left the hospital absolutely blind, with no perception of light whatever, the eye soft, and the folds of the retina close to the posterior surface of the lens. On my report, and the signature of a brother oculist, he received $5,000 from an accident insurance company some few weeks after he left my immediate care. I had fitted in the right orbit an artificial eye which caused some irritation, so from time to time he would seek me for its relief. He visited me for that purpose June 4, 1889, about one year after he left the hospital. When he sat down, facing the open window, I noticed that his pupil contracted. I shaded it and it dilated slightly, contracting again on exposure. Without speaking, I suddenly struck at his face with my open hand, so that its shadow fell upon the eye. His face and lids remained still, and he gave no indication whatever that he knew my hand was near him.

I inquired if he saw light. He answered, no : that it was like the darkest night to him ; and at my earnest solicitation repeated and added that he would be too glad to welcome light to deceive me, and hoped because his pupil responded to light I would not flatter him with unwarranted promises.

With the ophthalmoscope I saw that the retina had returned to its normal position over nearly the entire field; that the disc and macula were natural in all respects; and that the only evidences of disease were the remains of a detachment and marked choroidal changes at the outer and under segment of the equator. The tension of the eye was normal. I could see no reason why he should not regain some of his vision, and told him to note if in the future he should perceive any light. He returned in a month and reported that he could distinguish light from darkness; that at times he could make out the position of a window; that the world seemed to him as enveloped in a black cloud, through which, if it would grow lighter, he might distinguish objects. The cloud did grow lighter, it became more and more so every day; so in a short time he could easily make out the windows, the glare of the sun on the carpet, and any thing that stood between him and the light; then did the cloud seem to move away from before him, and as it retracted the lower field of vision came in view, objects became more distinct, especially about his feet. A few weeks after the first glimpse of light, he walked the streets alone. To-day, November 30, 1889, he goes unassisted over the city, comes to my office on the third floor of a large building, reads the signs in the streets with ease, and recognizes his friends when near. If it were not for a slight posterior polar cataract he could, with a glass, read large type.

The Present Condition of the Eye.—Externally it is normal to all appearances. Tension normal; Pupil round, reacts to light and shade; anterior chamber clear and of the proper depth, lens slightly opaque at its posterior pole. The cataract seems to be clearing up. With the ophthalmoscope, both by direct and indirect examination the following is seen:

The fundus of the eye is normal; the disc well marked; vessels, both arteries and veins, distinct and perfect; the color of the nerve pink, but not more so than we often see in healthy eyes. No pathological changes in or about the macula. Extending from the site of the wound, over an area which includes the entire subciliary zone, reaching to the external inferior border of the interocular field, is a large, black, white and red, mottled patch of choroiditis, over which can be seen the curved shadow of the detached retina.

At the site of the injury there is a bright star-shaped cicatrix. It is like a cross, one ray, the longest, reaching towards the macula; two others, of equal length, extending around the eye parallel to the equator; and one short club-shaped prong radiating towards the ciliary body. There are no deposits of pigment in or about this scar, which makes it probable that it marks the lines of rupture in the retina. On the opposite side of the eye is another well-marked glittering band in the retina. It is linear, running from the edge of the choroidal patch above described upwards and backwards as far as the equator, where it disappears in a fading point.

The above case is remarkable in several respects, which I shall only mention and not discuss. Before doing so, however, it is important to prove, to the best of my ability, that the patient was not an imposter, considering the large accident insurance policy he held. There can be no doubt that the detachment of the retina was the cause of the blindness. Three weeks after he was injured the eye was clear and vision most excellent. At that time the retina left the choroid, and as it raised up I could distinctly see its floating folds, marked by the retinal vessels. It finally closed in on the field of vision from both sides, so that in a few days the disc disappeared. Such could not be the history nor the picture of a dense opacity in the vitreous. That the patient was totally blind seems equally certain. He was a gentleman whose word was reliable; besides he developed a remarkable degree of tactile sensibility, so that he could read the elevated print and write with the sharp stylet. From the moment he saw the light this sense rapidly left him, and in two weeks he lost the result of a year's training.

In conclusion I present the following points for consideration:

1. The immediate cause of the detachment: did it take place before or after a rent in the retina?
2. The apparent disorganization of the eye, viz., loss of sight, reduced tension and the polar cataract.
3. The spontaneous cure of the detachment.
4. The return of the interocular tension to the normal.
5. The reaction of the pupil to light and shade, when there was no conscious perception of light whatever.

6. The return of the visual sense and the great extent of the visual field, which proves that the retina does not always degenerate or lose its function when detached.

7. I am inclined to think that the opacity at the posterior pole of the lens is less dense than when I first observed it. This, too, may disappear.

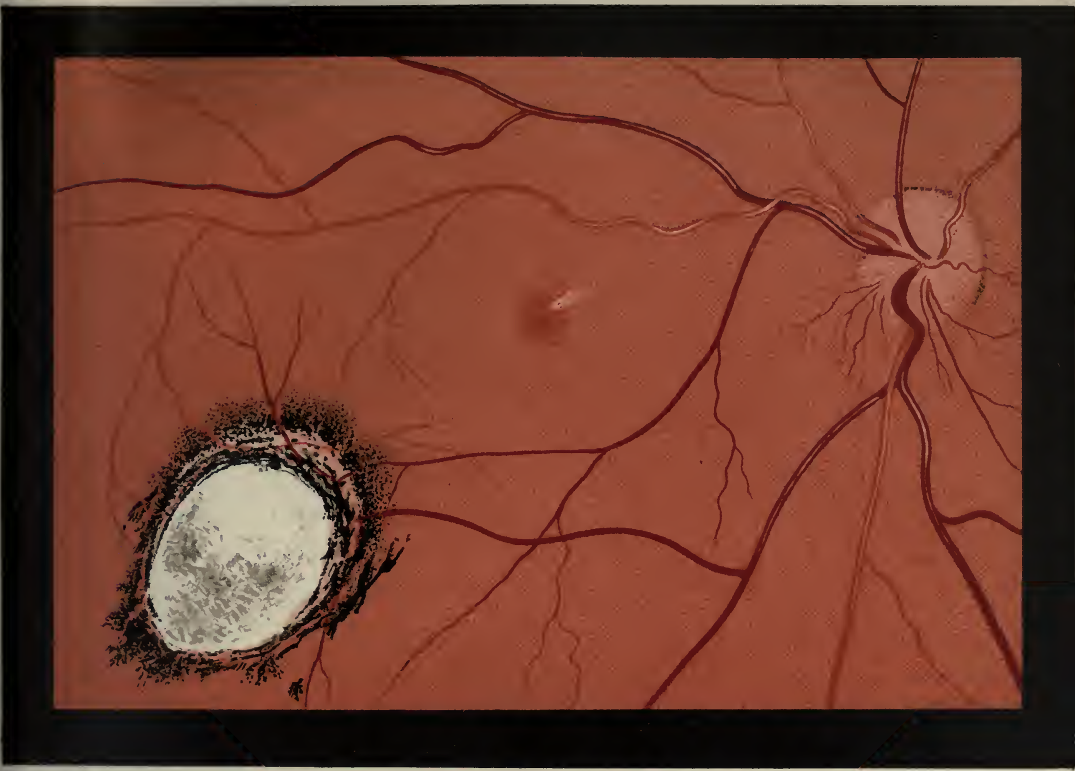

Fig 1.

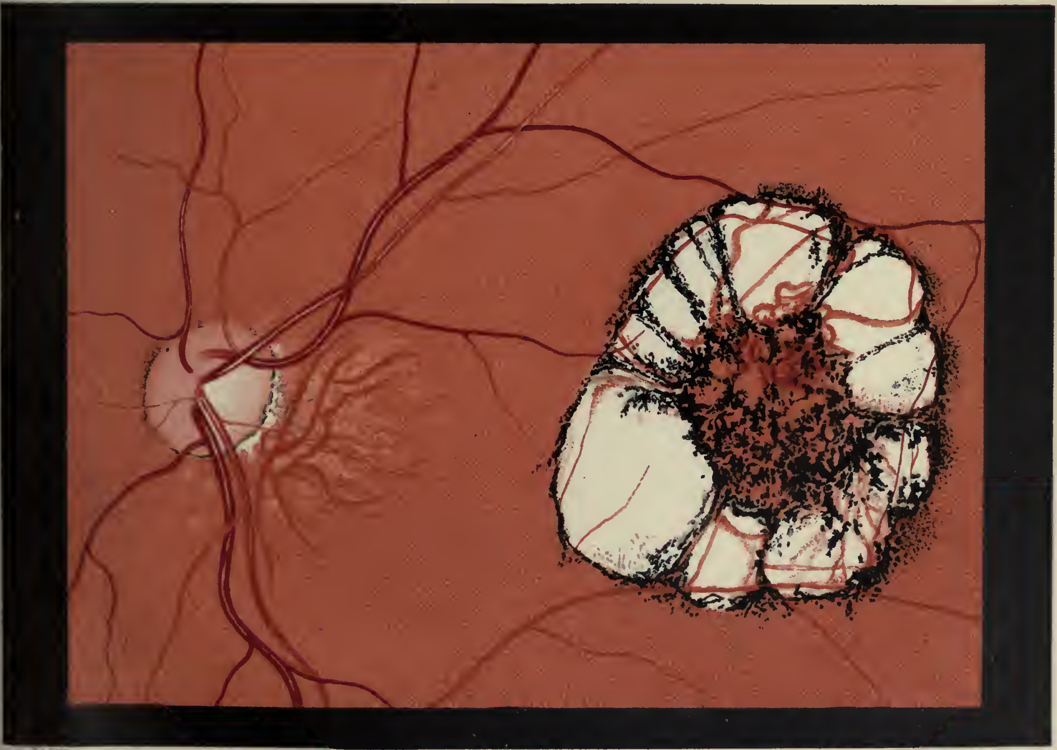

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Fig 2.

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Tab II

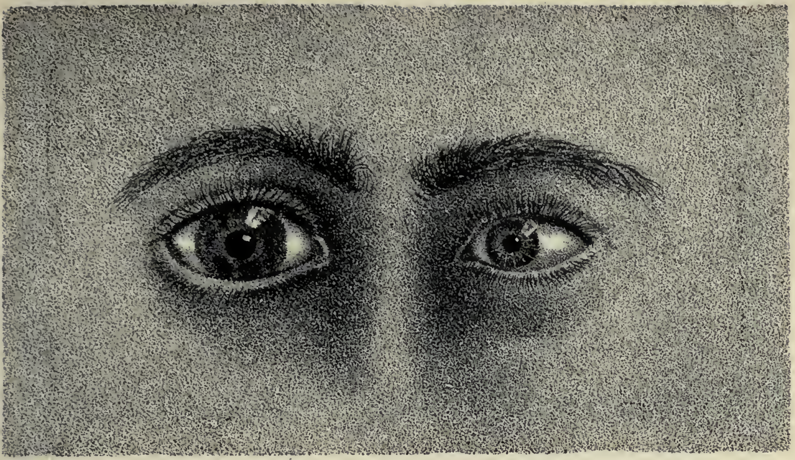

Fig 3

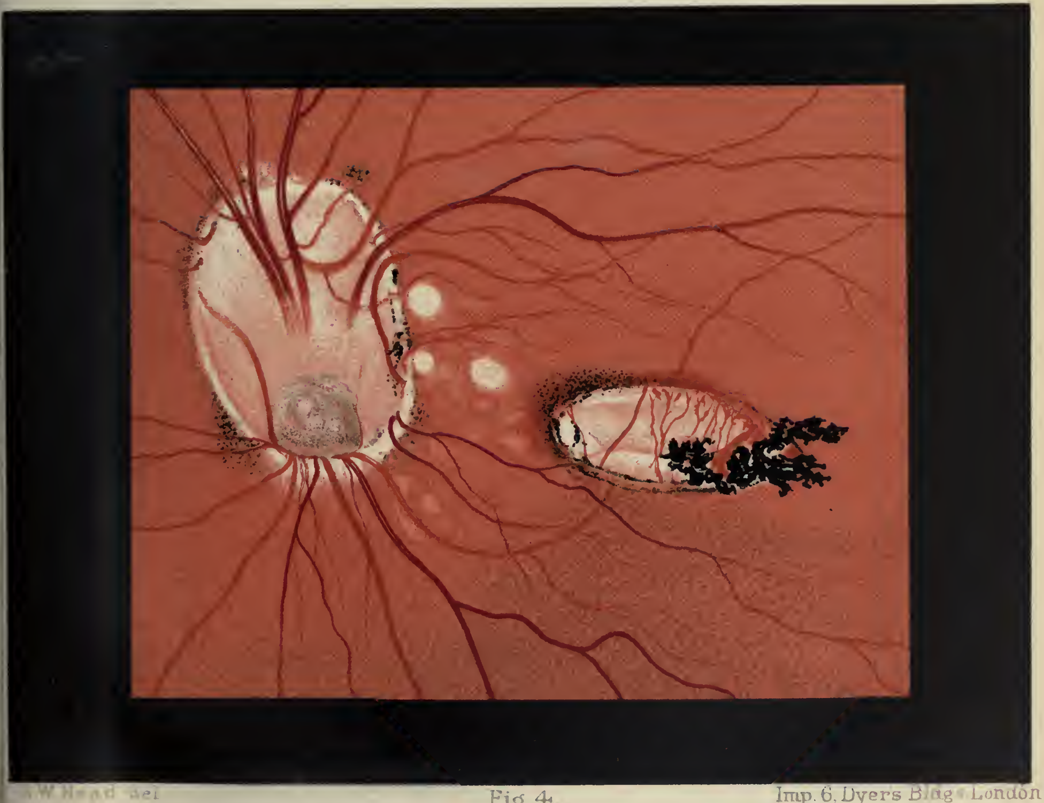

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Fig 4.

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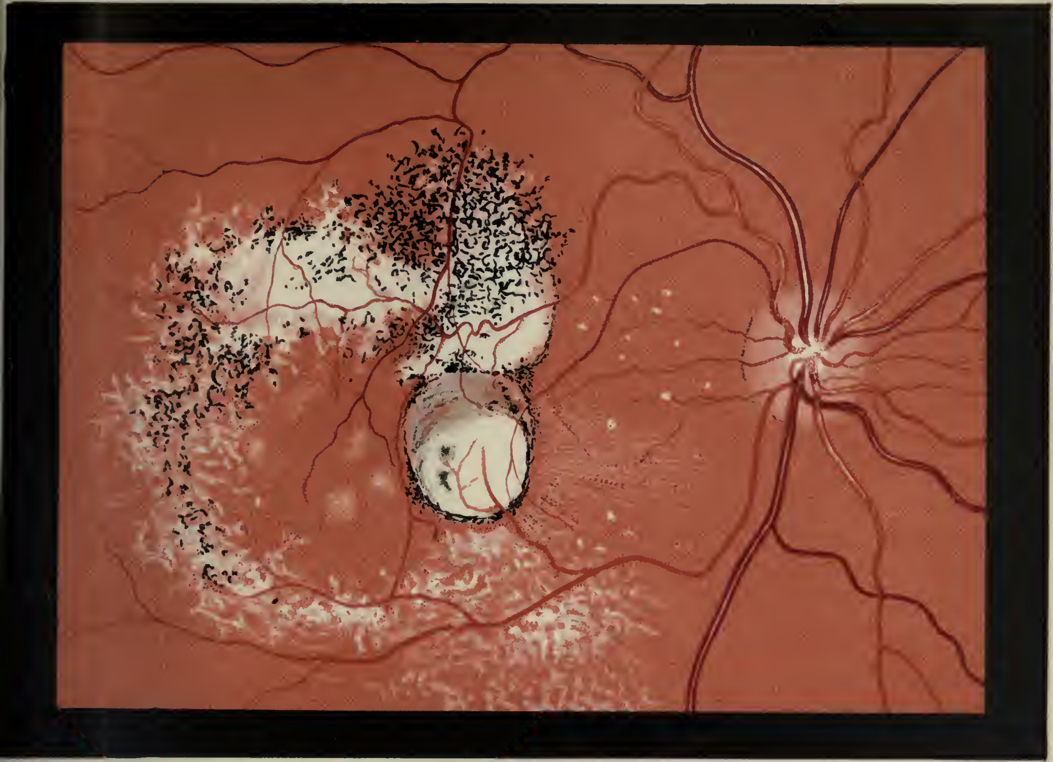

Fig 5

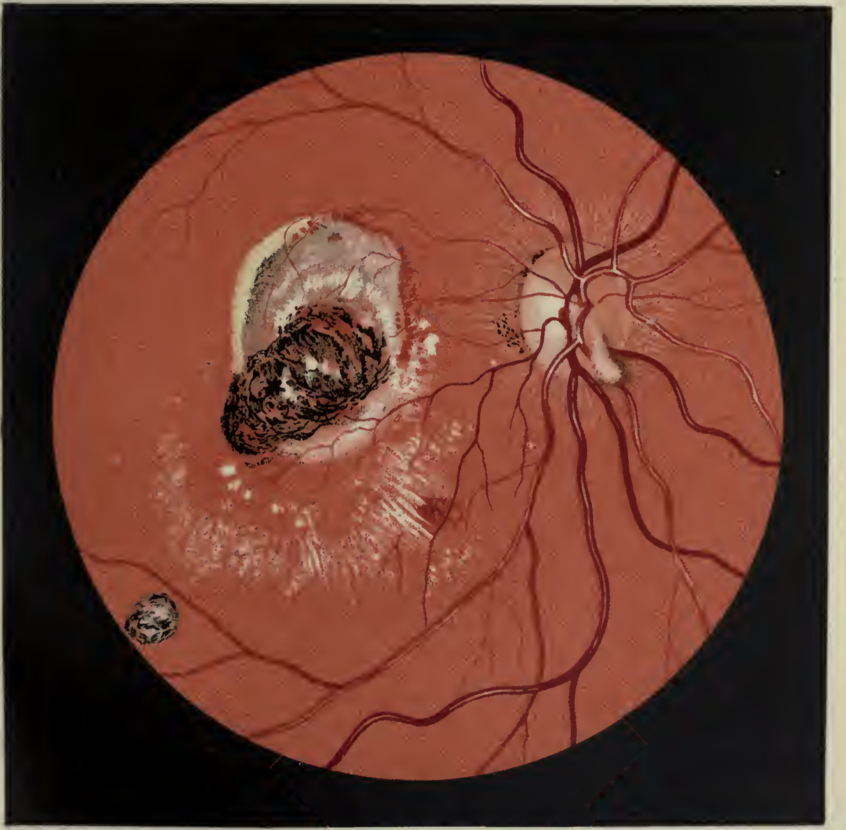

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Fig 6.

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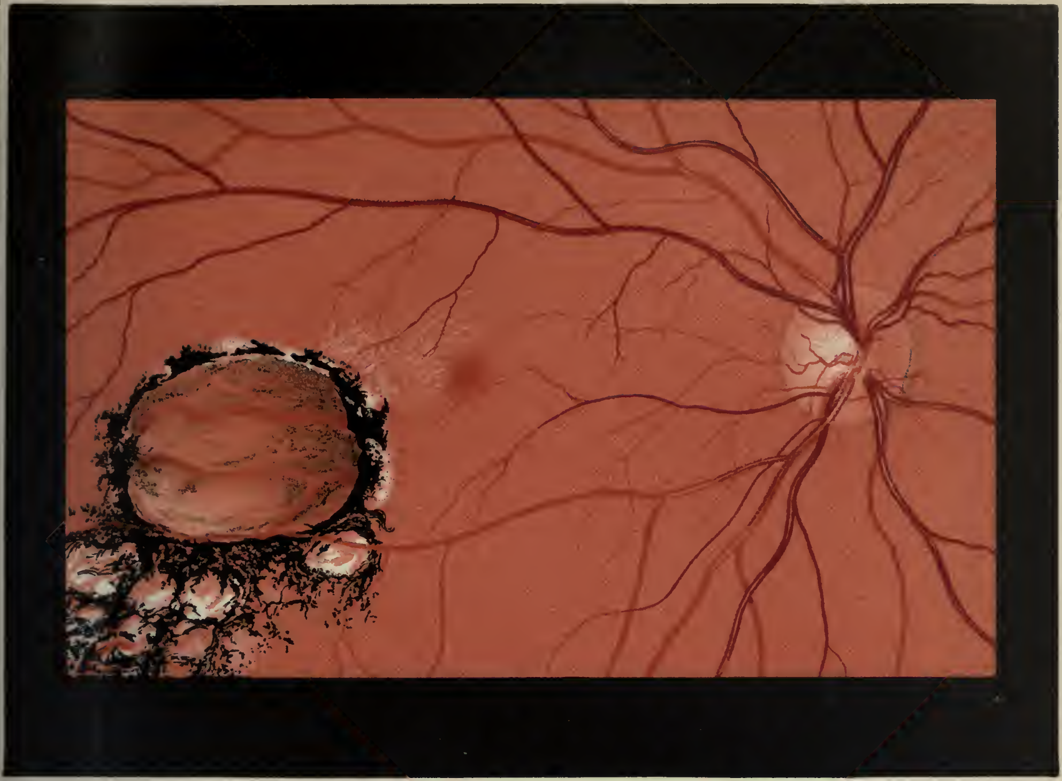

Fig 7.

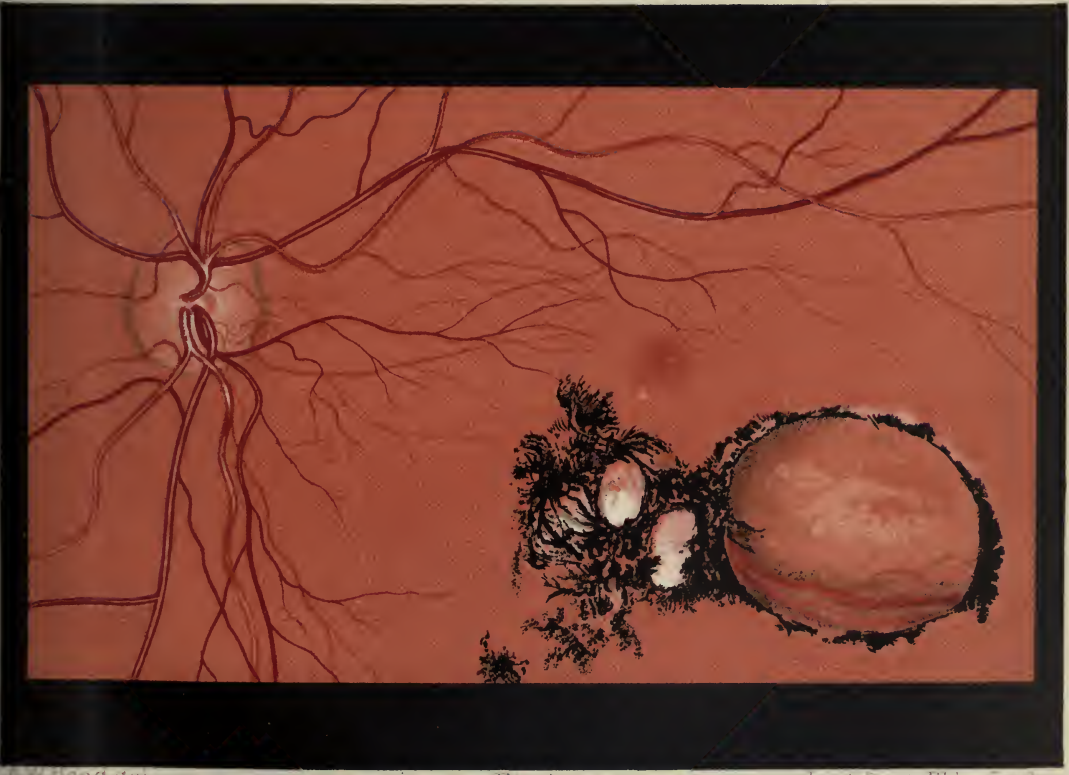

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Fig 8

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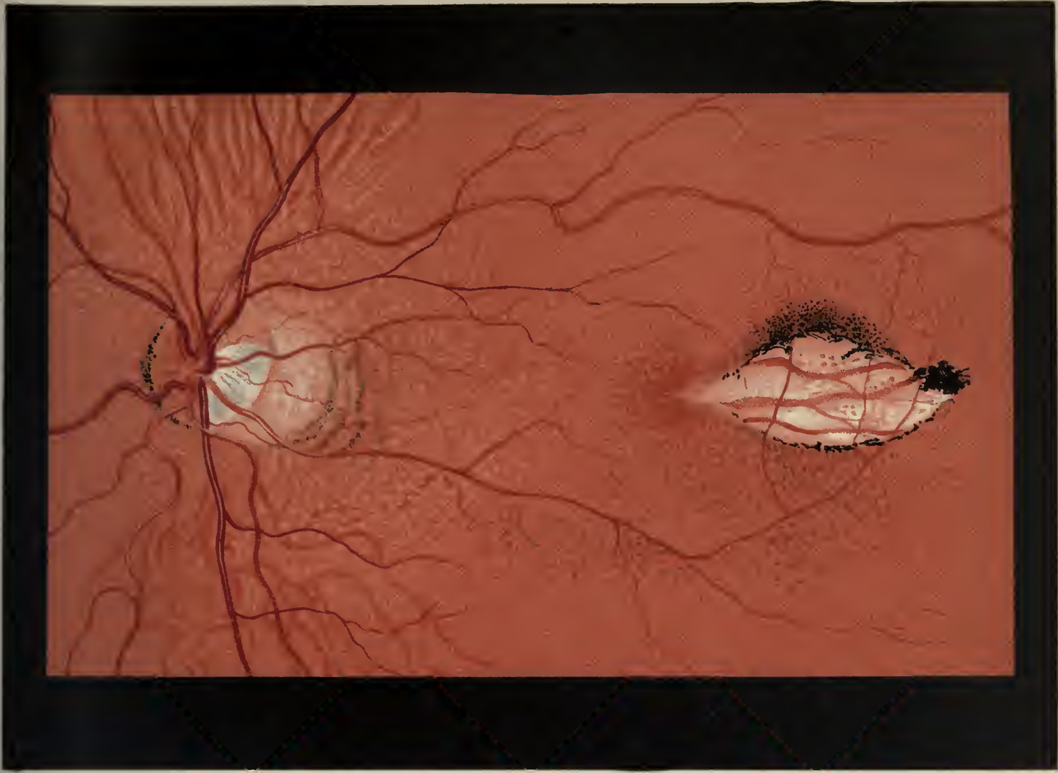

Fig 9.

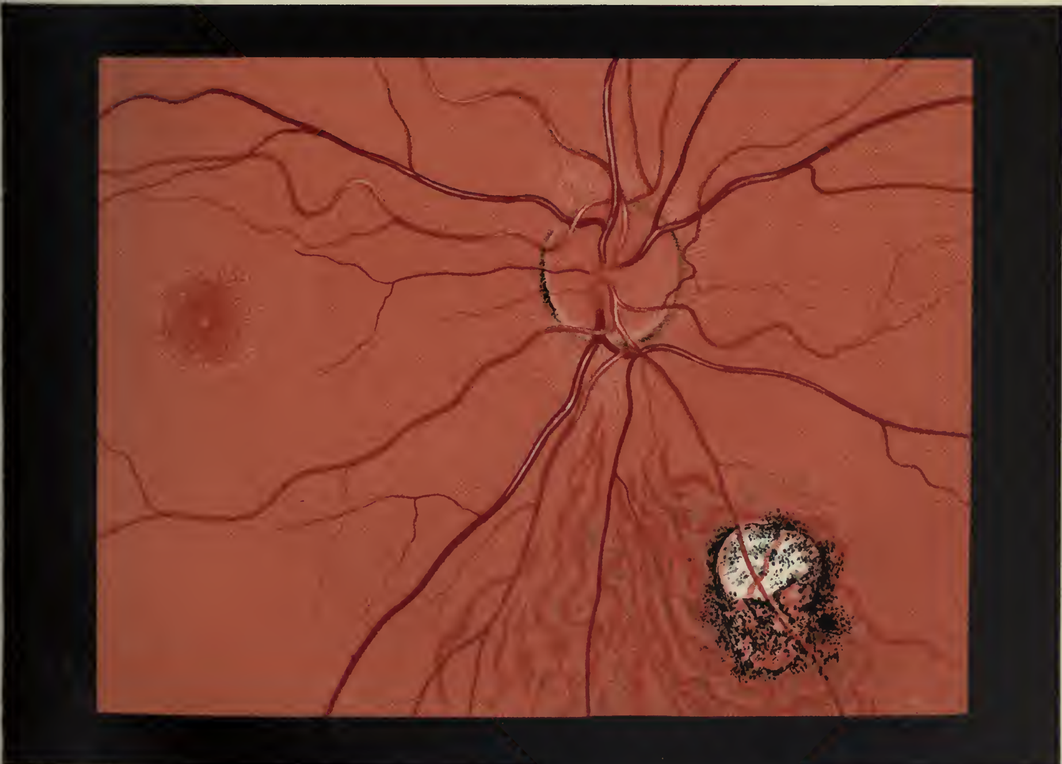

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Fig 10.

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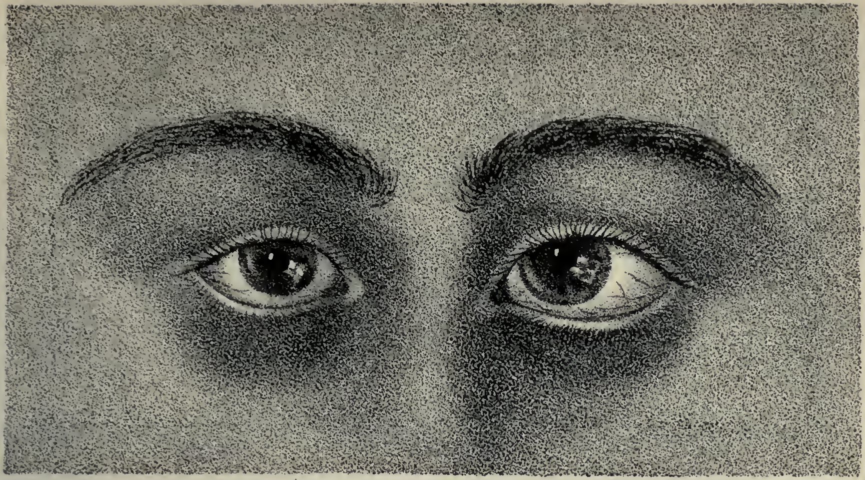

Fig 11

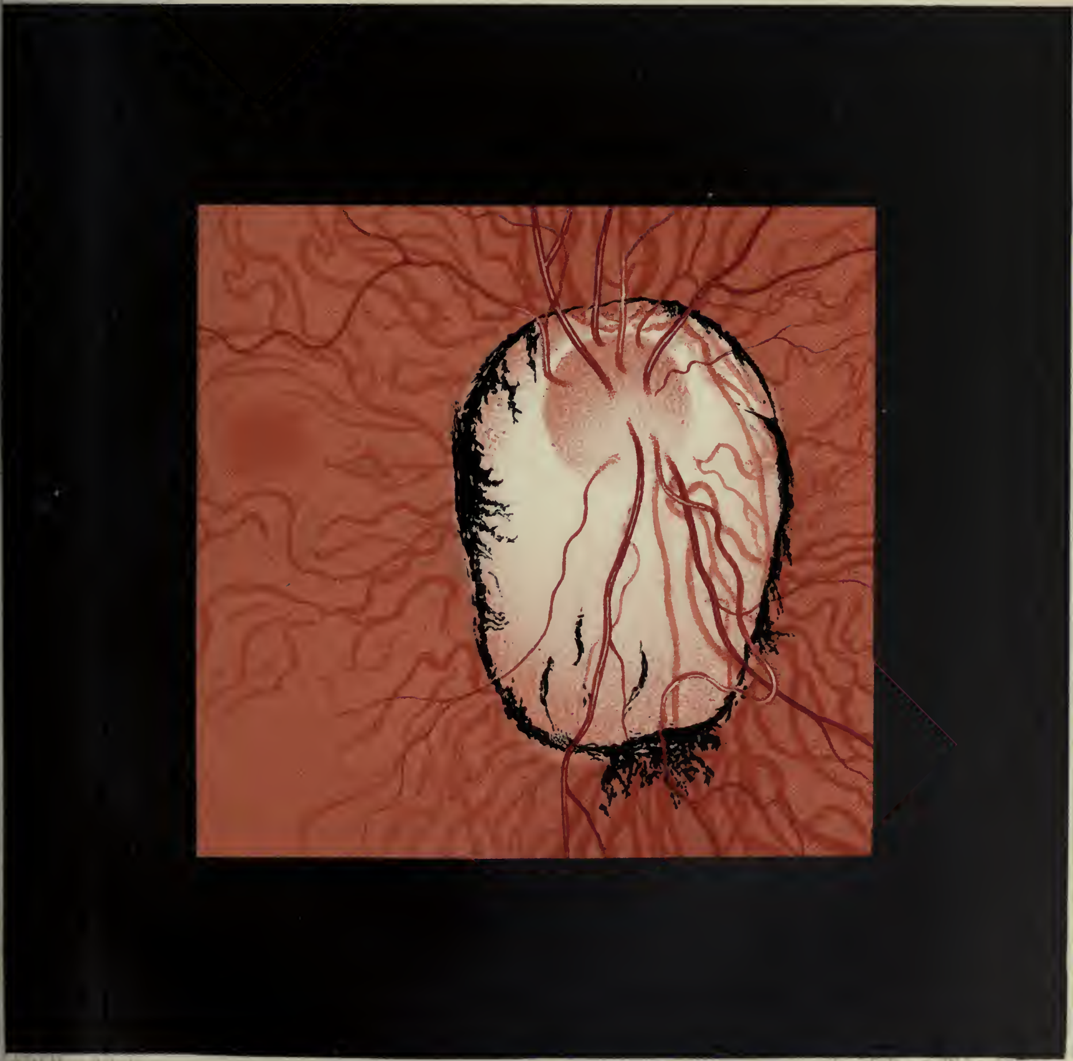

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Fig 12

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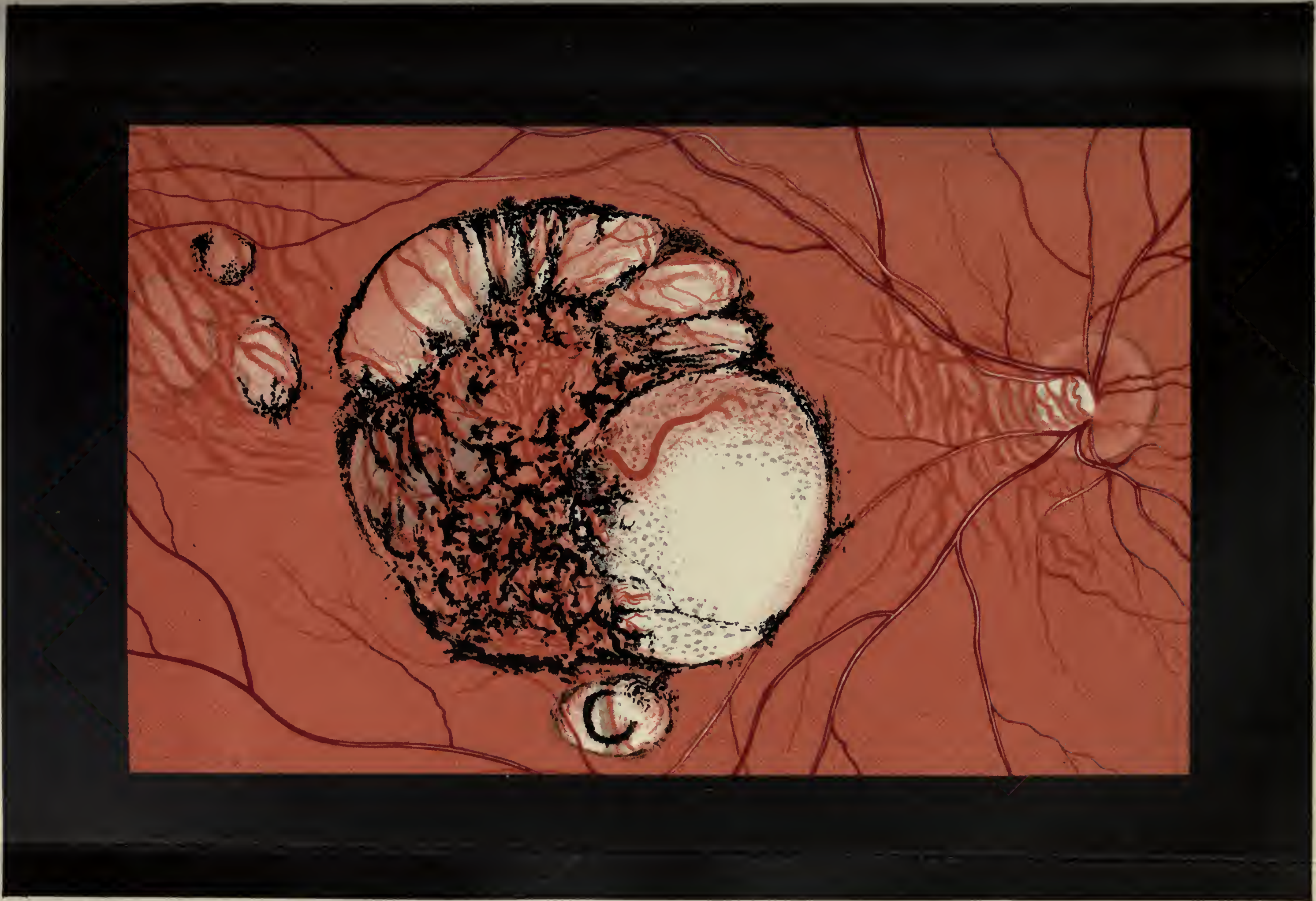

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Fig 13

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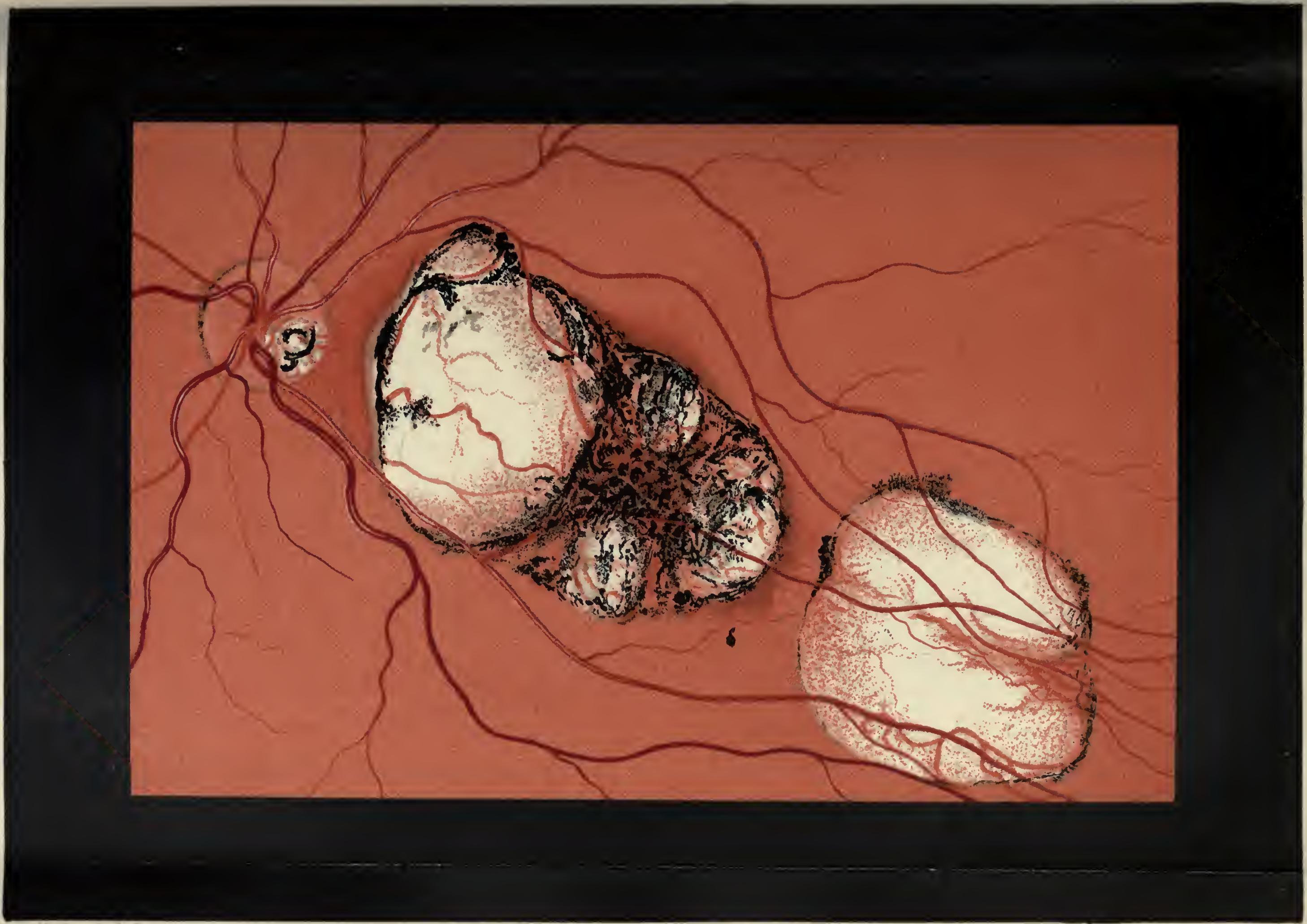

A W Head del.

Fig 14.

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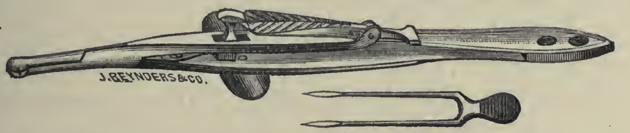
ON THE EXTRACTION OF LENSES DISLOCATED IN THE VITREOUS.

By H. KNAPP.

(*With a wood-cut.*)

AT the meeting of the American Ophthalmological Society in July, 1885, Dr. C. R. Agnew communicated a case[1] in which he had extracted, by means of a double needle, *bident*, a lens dislocated into the vitreous of a blind but otherwise not disfigured eye. "The lens was so loose that it moved to and fro, though suspended, on the temporal side, by a narrow, hinge-like attachment of the suspensory ligament." Dr. Agnew describes the operation as follows: "I penetrated the temporal aspect of the eyeball at a point just far enough back to enter the vitreous chamber without wounding the iris or touching the dislocated lens, till the bident had penetrated the vitreous humor to a point a little to the temporal side of the centre. I then, by depressing the handle of the holder [a Sands needle-holder], caused the points of the bident to describe an arc forward. I was greatly pleased to see the lens caught by the bident—as a pea might be lifted on a two-tine fork—and brought forward through the pupil into the anterior chamber. I then pushed the bident on and caused its points to emerge on the nasal side of the eyeball close



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[1] Transact. Am. Ophth. Soc., 1885, p. 69, etc.

behind, but avoiding the iris. . . . With a narrow Graefe's knife I then made a sufficient lower corneal section and easily completed the delivery of the lens by a delicate wire spoon. I then removed the bident, . . . without difficulty. There was scarcely any escape of fluid, the corneal wound fell into neat coaptation, and there was no prolapse of iris. . . . The recovery was uneventful; the cure was complete in seventeen days. The pupil had become a little drawn in the direction of the corneal wound, though contractile and not adherent."

This ingenious operation has since been practised at the Manhattan Eye and Ear Hospital. Dr. D. Webster[1] communicated another case at the same meeting of the Society. At the meeting of the Society in 1888,[2] Dr. O. D. Pomeroy detailed six cases, four from his own practice, one by Dr. Agnew, taken from the case-book of the Manhattan Hospital, and one from Dr. Webster. Dr. Webster mentioned that Dr. J. A. Andrews had also made the operation three or four times. Dr. Pomeroy stated (*l. c.* p. 172) "that a modification of the instrument has been made by Dr. J. A. Andrews of New York, in which the handle is fastened on by a delicate spring, admitting of much more nicety of manipulation than when the Sands needle-holder, of the original bident, is used. The handle should certainly *not* be reapplied to remove the instrument from the eye, the fingers of the operator allowing of the removal in a much more delicate and safe manner."

I must confess that I have not been favorably impressed with Dr. Agnew's bident operation. The reason is, that in my practice I have always been able to remove such lenses without a bident, and with no greater accidents than are recorded in the above bident cases. Yet the extraction of dislocated lenses may offer unusual difficulties, and such cases are not so frequent as to warrant any one in declaring an instrument like the bident to be always superfluous; I personally am not prepared to say that I shall never use it. I want, in the following pages, to express my own views and experience. The value of an operative method can only be decided by the comparison of parallel cases, and therefore I beg leave to introduce my remarks by the record of a case which was as exactly alike to Agnew's first as two cases can be. I can assert this positively, for I knew Dr. Agnew's case not only from his description, but by personal examination, the patient having for years consulted Dr. Agnew and myself. Dr. Agnew adds to the communication of his case the following remarks (*l. c.* p. 76): "It may be said that the lens might have been removed by using a large spoon through a section of the cornea with or without iridectomy. That with other methods was considered and dismissed as impracticable. I had the case under observation for six or seven years and studied it in the light of every known surgical procedure. I do not think that I could have met the indications with any of the methods previously in use." That this might have been not only possible, but feasible and easy, without resorting to any extraordinary operative procedures, will be demonstrated by the following case.

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[1] Trans. Am. Ophth. Soc., 1885, p. 76.

[2] Trans., 1888, p. 168.

Sept. 30, 1889, I was consulted by Antonio B., a Mexican of 26 years of age, who stated that he did not remember ever having had sight in his right eye. For 13 years a white body had been noticed in his right pupil. The left eye had ever been excellent, and both eyes free from irritation until the last month, when the right eye had occasionally been painful and red. At the time he presented himself I found the left eye normal in every respect, the right normal in size and tension. Behind the pupil, which moved consensually with the other, a whitish-yellow lens, about two thirds the size of a normal lens, floated to and fro with every movement of the eye, disappearing from sight when he reclined, occupying the pupil when he bowed. It even was alleged that the cataract at times protruded through the pupil, but I have not been able to produce this effect by letting him bend his head forward. The eye was completely blind, and the whitish retina could be seen by oblique illumination.

As the eye showed no special degeneration, in particular no irido-cyclitis, I advised the patient to have the cataract removed, thinking that the floating lens, after a period of rest, had now become troublesome and would probably continue so. The patient consented to my proposition so much the more readily as he expected from the operation not only the subsidence of his recent discomfort, but also the removal of the disfigurement. I told him I trusted that the operation would succeed, but it might fail, as all operations occasionally did, and in this case the removal of his eye might be necessary.

The operation was made at the N. Y. Ophthalmic and Aural Institute, Nov. 16, 1889, as follows. After cocaine had been instilled, I let the patient lie on his face, to induce the lens to enter into the anterior chamber. This not being obtained, I let him sit on the operating chair and instilled cocaine several times more, cleansing and sterilizing the lids and eye as before the extraction of an ordinary cataract. When the eye was perfectly anæsthetic, showing, however, only a minimum dilatation of the pupil, I applied the ordinary wire speculum, steadied the globe by grasping the anterior part of the lower episclera with a pair of fixing forceps, made a section with a Graefe knife through the upper part of the cornea, the edges in the transparent margin, the apex 2 *mm* below. I removed the speculum, held the upper lid with the index of my left hand, and pressed with the thumb of my right the edge of the lower lid on the lower part of the sclera, directly toward the centre of the eyeball. At once the lens presented in the pupil, and by pressing more firmly on the eyeball, always in the direction toward its centre, it was gradually driven through the pupil and into the gaping corneal section which the iris and lens completely filled. When about two thirds of the lens were in front of the wound, Dr. Born, who had stood in readiness for this manœuvre, lifted with a wire spoon the lens completely out in its unbroken capsule. I released the lids, the iris fell back spontaneously, and there had been no escape of vitreous during the whole procedure. The eye was bandaged and the patient put to bed. The healing was undisturbed and perfect. No trace of anterior or posterior adhesions, the pupil round, central, and consensually movable. The patient was discharged in two weeks. Four or five weeks after the operation I presented him at the ophthalmological section of the N. Y. Academy of Medicine, where the president, Dr. O. D. Pomeroy, though unwilling to generalize the method, declared that nothing could be neater than this result.

I admit that this was a most felicitous operation; but it shows that it is possible, by external manipulation to extract lenses dislocated and swimming in the vitreous and to get them out without accident and with preservation of the natural pupil.

Another more numerous group of cases, which can be dealt with in the same manner, are half-dislocated (subluxated) lenses, of traumatic origin or after degenerative processes,—the tremulous, hypermature, and displaced cataracts. They are often enough congenitally dislocated to a small extent, and become more so in the course of years. As long as they remain transparent and quiet, they should be left alone, the same when they become opaque, but leave a sufficient space of pupil free, or show a tendency to further displacement; but when they become totally opaque and occupy the whole pupil, they can either be extracted or left in the eye, and the pupil dilated by an iridectomy. Also the dislocation of the cataract which sometimes occurs during an ordinary extraction, frequently with prolapse of vitreous, can appropriately be treated in the same way. I have had representative cases of all these conditions. One recent example may suffice to illustrate the whole group.

Philipp H., German, forty-nine years old, received a blow on his left eye, dislocating the lens down and outward. The pupil was dilated, the nasal half of the iris paralyzed. The lens at once became cataractous, but the eye remained irritated. Four weeks after the accident, on September 18, 1889, I extracted the lens in its capsule, with some prolapse of vitreous, without iridectomy, by external manipulation, as described in the above case. Recovery undisturbed. Patient discharged in twenty days; opacities of the vitreous and the partial dilatation of the pupil still present. S $\frac{20}{70}$, when discharged. Patient has been seen since. Eye free from irritation. S $\frac{20}{50}$, Dec. 28, 1889, though fine cords ominously stretch from the nasal end of the scar toward the temporal side of the ciliary body.

This method of operating has been general with me for many years, and I am sure that there was no case in which I have left the lens in the eye.[1]

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[1] In all my practice I have only once been obliged to leave an extraction unfinished. It was in a woman, who had so extensive a symblepharon, with pannus after old trachoma, that I could not expel the cataract through the small, freely bleeding section, the opacity of the cornea and the blood in the anterior chamber making further steps too uncertain. The patient has not reappeared.

Though almost always it is possible to extract the lens by external pressure, I have, in every case of extraction, a sterilized wire spoon and a broad metal spoon at hand, but for more than ten years I have not been obliged to make use of these or any other traction instruments. When a dislocation of the lens in a blind eye is complicated with irido-cyclitic processes, I advise enucleation.

In the case of Dr. Agnew, from which I quoted so extensively in order to acquaint the reader of these ARCHIVES with the necessary details, if they desire to repeat the operation, I advised enucleation, although there was no iridocyclitis. I did this under the influence of a case in which, shortly before, I had removed a dislocated lens by external manipulation without much difficulty. There was no iridocyclitis, but the lens was attached rather firmly to the lower part of the ciliary body; the recovery had been slow, and the eye had remained tender for months. I reproached myself with having let the patient run great risks for little gain. I am glad that Dr. Agnew took a more favorable view of his case, and am happy that the operation has proved a permanent success. This will be seen from the following statement which Dr. Webster was kind enough to send me on my enquiry about the further condition of the patient: "I have seen him many times since. I saw his father this last September and enquired about him. He is married since, and is the father of a happy family. His eye gives him no trouble, and he constantly rejoices that he is not wearing a glass eye."

I am averse to the bident, because its use is incompatible with the methods of external manipulation, methods which, thus far, have served me well. When an eye is once transfixed with that instrument from one side to the other, the natural expulsion of the lens by spontaneous intraocular or artificial outside pressure is out of the question, instrumental delivery becoming a necessity. Even this has not always been successful, for, in some cases, the bident had to be withdrawn in order to make the extraction of the lens possible. I should, perhaps, not criticise an operative method which I have never seen, for it may in reality be different from what I image it to be, but, apart from the above objections, it seems to me that the last step of the operation, the removal of the bident, must be very unpleasant, though Dr. Agnew says it was easy, and the records of the other cases only hint at difficulties. In any case of extraction when prolapse of vitreous is either present or imminent, I consider it prudent at once to remove the speculum and complete the operation without pulling at the eyeball. In the bident operation, when the lens is out and the vitreous chamber opened, a two-pronged fork, piercing the sclerotic in four places, has yet to be drawn out. Tempting as Agnew's operation is, I shall reserve it for exceptional cases, for it is complicated, and contends with difficulties which, I think, do seldom exist. Unless peculiar conditions make a change of plan desirable, I shall, for the extraction of dislocated lenses, operate as before, viz.: Thorough local anæsthesia, upper corneal section, removal of the speculum, expulsion of the lens by methodical external pressure. If I fail I shall introduce a wire or metal spoon through the corneal section and the pupil, and extract the lens in this way. The necessity of introducing a spoon is, so far as my experience goes, very rare.

ACUTE MANIA FOLLOWING THE ENUCLEATION OF AN EYE.

By W. CHEATHAM, M. D., Louisville, Ky.

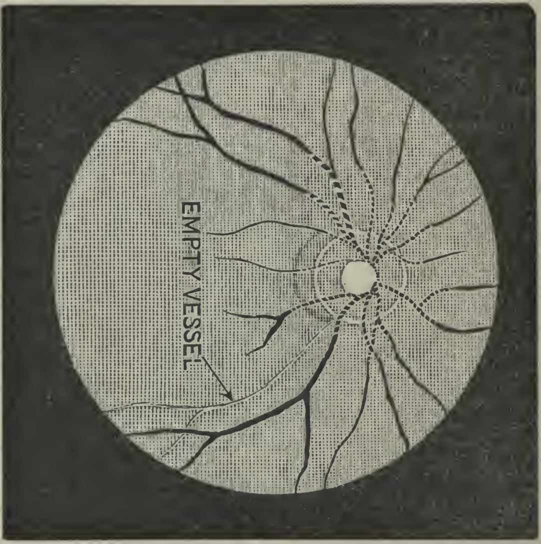
F. B., age thirty-five, carpenter, was struck in the left eye, on September 30th last, by a spike six inches long, rupturing the eye at the inner sclero-corneal junction, and also at the insertion of the external rectus. I saw him late in the afternoon of October 1st. The whole organ was greatly inflamed, the chemotic conjunctiva, even at this early stage, overlapping the cornea. I advised immediate enucleation, which was done early on the morning of October 2d, quite a good deal of hemorrhage following. The instruments were washed in boiled carbolized water, the sponges put in boiled water and hyd. bi-chlo., and the eye dressed with iodoform. The spike he was struck with was very dirty and rusty, and the patient fell on his face in the dirt when he was struck ; he was brought home with a dirty cotton handkerchief of a fellow-workman over his eye. October 3d there was great swelling of the parts, with great pain, and the patient was controlled with much difficulty ; he imagined he was from home, and some one wished to force him to join the church. Morph. sulph. was given him hypodermically and by mouth. October 5th he was very violent ; the parts were very tense. Pot. brom., in large doses, was added to the morphine. He would allow no local applications to be made. Drs. Mason and Cartledge were asked to see the patient with me. Thinking the morphine had something to do with his present condition, bromidia was given in teaspoonful doses, every one, two, or three hours, until October 8th. This failing to quiet him, the morphine had to be used again occasionally. Sometimes it required four or five men to hold him in his bed. He recognized no one ; endeavored to do violence to his wife. October 13th I gave him hydrobromate of hyoscine, gr. $\frac{1}{100}$ four times a day. Just at this time the inflammation began to subside and his mind to clear up. During his illness his pulse and temperature were both normal, except the afternoons of October 10th and 11th, when the former was 96 and the latter $99\frac{4}{5}$°. The patient was discharged October 20th well.

A PECULIAR AFFECTION OF THE NEURO-RETINAL CIRCULATION.

By W. CHEATHAM, M. D.

(*With a wood-cut.*)

May H., age fifteen, a rather well-developed girl, anæmic in appearance, presented herself October 17, 1889, with the following history : On awaking on the morning of October 17th she discovered she was blind in her right eye. She has had frequent attacks of so-called follicular tonsillitis, having just recovered from a severe attack. When sick reads lying in bed ; has done a great deal of it lately. She has never menstruated. V : R = 0, L = $\frac{20}{50}$, with a + $\frac{1}{60}$c axis vertical = $\frac{20}{20}$. The nerve and retina present such an appearance (except the vessels) as seen in embolism of central artery of retina. There is extensive exudation into nerve and retina, with a cherry-red spot at macula lutea. The retinal vessels present a very peculiar appearance. The arteries near the nerve were normal in color and size ; some of them about the equator of the eye appeared plugged with very dark blood ; one branch given off from a main artery below was entirely empty ; most of the arteries on the nerve, and for a short distance off, had white streaks along each side. The veins from the periphery to the equator were filled with clotted blood, as the arteries were, so it was difficult to distinguish between them. A piece of the clot, about two or three lines in length, would break off from the large clot in the vein above, wash down some distance, disappear under a small exudation, and appear on the other side as a fine dust or powder, carried by a yellowish fluid, and washed out by this fluid into the central vein. As this piece of clot disappeared, the large clot did not apparently decrease in length, so it must be slowly moving. Most of the veins below had apparently no current, but were filled with small clots as they neared the optic nerve. I ordered Flexner's syr. albuminate iron, with quinine and strychnia, and pot. iod. There was no heart complication. Mr. J. A. Flexner examined her blood in my office, and found the corpuscles normal in quantity, but carrying only 67 % of hæmoglobin. He also found her urine deep yellow in color ; sp. gr. 1,015 ; no albumen ; oxalate calcium present ; Indicans and pus present ; reaction acid ; sediment abundant ; sugar, uric acid, phosphates, bile, blood, and mucus absent ; pavement-epithelium abundant ; hyaline casts present, but not numerous ; urea only 1.6 %.



October 18th.—The exudation has increased, the outlines of the nerve very much less distinct, and the cherry-red spot at macula is nearly gone ; the retinal circulation is better ; the artery below, which was better yesterday, now fills every few minutes, then again empties ; both arteries and veins pretty well filled, but the current can still be seen in the veins flowing slowly, and now the current in the arteries can be seen distinctly. No pulsation in either arteries or veins ; the clots in the extremities of the vessels are nearly gone ; the blood in both veins and arteries appears finely granular.

October 19th.—Exudation still increasing ; current in vessels can still be seen ; artery, which was empty, now full.

October 21st.—Exudation increased ; two small hemorrhages near macula, vessels all full, and no current discernible.

October 23d.—Exudation much less. R V = 0. L V = $\frac{20}{20}$, with correcting-glass.

November 20th.—Nerve and retina atrophied.

This case does not appear to me to have been embolic in character in the beginning, but more like one in which a diseased condition of the vessels themselves was the original cause; it answers more closely to some of the series of cases reported by Dr. Ole Bull, under the title, "Pathological Alterations of the Retinal Vessels," at the Ninth International Medical Congress.

A METRIC SYSTEM OF NUMBERING AND MEASURING PRISMS.

By CHARLES F. PRENTICE, of New York.

Introductory Remarks by Dr. Swan M. Burnett.

["The old method of numbering prisms simply by the angular deviation of their sides is, confessedly, inaccurate and unscientific. Any attempt to supplant this by one more accurate, and to place the nomenclature of prisms on the same basis of scientific exactness as the other optical appliances in the hands of the practical ophthalmologist is, therefore, deserving of consideration. The method proposed by Mr. Prentice, in the following paper, not only does this, but does it in a manner and according to principles which are familiar to even the less scientific practitioners. To have the same unit (the meter) of measure and comparison for all refracting apparatus and uniform with the nomenclature employed in the designation of anomalies of refraction and muscular equilibrium, gives a simplicity which is not only commendable in itself, but tends to render the study of the practical use of prisms easier and more comprehendable to the student. This is particularly apparent in the connection the author establishes between the prism-dioptry and the meter-angle. Not the least important part of the contribution is the description of the instrument Mr. Prentice has devised for illustrating his idea and for testing the refraction of prisms generally. Such an instrument has always been a desideratum, and Mr. Prentice's well-known technical skill, combined with his thorough knowledge of mathematical optics, are a sufficient guaranty that these instruments, which are made under his personal supervision, will fulfil all the requirements of the case."]

THE present method of designating prisms by the angular deviation of their refracting surfaces, is open to the objection that we thereby define only an isolated feature of their construction, to the utter disregard of the varying powers of refraction, which must result from the use of refracting substances having different indices.

With a view of securing greater accuracy and uniformity in our utilization of the refractive properties of prisms, the following system of numbering, which I believe to be feasible, as well as suited to the requirements of ophthalmologists, is presented.

Let *abc*, Fig. 1, represent a prism, with the ray *i* incident perpendicularly to *ab*, and we shall have dv as the deflection accruing from the refraction at *e*. Similarly, dV will represent the deflection arising from the refraction at the same point *e*, for a prism, *ABC*, of greater angle.

We shall then have

$$dV : de = d_1v_1 : d_1e$$

But $$d_1v_1 = dv = \delta$$

$$\therefore \frac{dV}{de} = \frac{\delta}{d_1e}$$

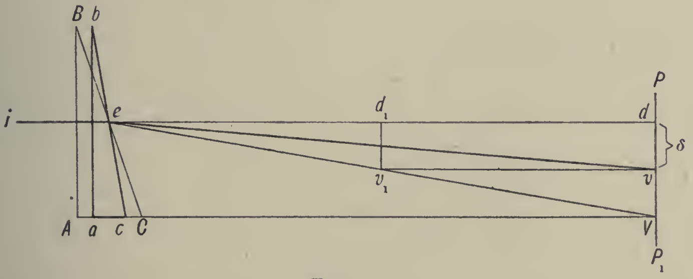


FIG. 1.

By adopting one meter as the distance of the point *e* from the plane PP_1, in which the amplitudes of deflection dv and dV are measured, we shall have $de = 1$, when

$$dV = \frac{\delta}{d_1e} \quad . \; . \; . \; . \; . \; . \; . \; . \; . \quad (1)$$

It is further evident that the greater the refractive power of the prism, the greater will be its correlative deflection in this plane, so that

$$R : dV = r : \delta \quad . \; . \; . \; . \; . \; . \quad (2)$$

where R and r represent the refractive powers of the prisms *ABC* and *abc*, respectively.

Consequently, $R = dV$, when $r = \delta$.

Substituting these values in the equation (1), we have

$$R = \frac{1}{d_1e} \cdot r$$

or, if $d_1e = x_m$ as a function of the meter,

$$R = \frac{1}{x_m} \cdot r$$

Allowing r to represent the unit of prismatic refraction, we then have

$$R = \frac{1}{x_m}$$

Consequently, the prismatic refraction is in inverse proportion to the distance at which the unit deflection is produced, being fully in harmony with the refraction of a lens, which is in the inverse proportion to the distance at which the image is formed.

Provided, therefore, a standard amplitude of deflection be adopted as the unit, and which shall be measured in a plane one meter from the refracting surface of the prism, we shall be enabled to designate prisms, in dioptries, for instance, with the same significance as in lenses.

Thus prisms of two, three, or four dioptries will produce the same unit deflection at one half, one third, or one quarter of a meter, respectively.

By the relation (2) we shall also find the same prisms to produce two, three, or four times the unit deflection *in the meter-plane*, so that the problem reduces itself to the selection of a series of prisms which shall produce tangent deflections, at this distance, which are multiples of the adopted unit.

As the deviation produced by a prism will be dependent upon its angle and the index of refraction, we may here note the relations existing between these factors, when, as in Fig. 2, a ray i is incident perpendicularly to the face ab, and, consequently, suffering refraction at e only. Here we have $< abc = \beta$, as the angle of the prism; $< iep_1 = \beta$, the angle of incidence; $< pev$, the angle of refraction; $< dev = \gamma$, the angle of deviation.

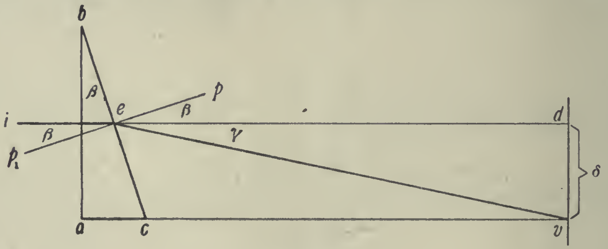


FIG. 2.

Consequently the index of refraction

$$\eta = \frac{\sin < pev}{\sin < iep_1} = \frac{\sin\,(< ped + < dev)}{\sin < iep_1} = \frac{\sin\,(\beta + \gamma)}{\sin \beta}$$

As the angles of prism which we shall generally have to consider are small, we need only allude here to the formula for the minimum deviation, which is

$$\eta = \frac{\sin \frac{(\beta + \gamma)}{2}}{\sin \frac{\beta}{2}}$$

A relation, therefore, exists between η, β, and γ, which requires that two of these factors be known to enable us to determine the third, so that our choice of a unit deflection will be included in the following propositions, wherein prisms of low degree, the usual limits of refractive index, and comparatively small deviations of the refracted ray would have to be considered :

Proposition I.—The values of η and β being given to find γ
" II.— " " " η and γ " " " " β
" III.— " " γ and β " " " " η

For purposes of illustration and reference, we may consider only the first proposition, for the generally accepted index, $\eta = 1.53$ (Spiegelglass), for a 1° prism, when we have

$$\frac{\sin\,(1^\circ + \gamma)}{\sin 1^\circ} = 1.53 \therefore \sin\,(1^\circ + \gamma) = 1.53 \times 0.017452 = 0.026701 = \sin 1^\circ\, 31'.8$$

$$\sin\,(1^\circ + \gamma) = \sin 1^\circ\, 31'\, 48''$$

$$\therefore \gamma = 31'\, 48''$$

The deflection δ, corresponding to the angle γ, being equal to

$$de.\ tang\ \gamma = 1_m\ tang\ \gamma = tang\ 31'\, 48''$$

we have, in meters, $\delta = 0.009250$.

A prism producing a deflection equal to the tangent of 31′ 48″, equal to 0.00925 at one meter distance, will therefore correspond to the accurately ground prism of one degree angular opening, and with an index of 1.53.

In case glass of another index were used, it would be necessary to vary the angle of the prism, so as to satisfy the conditions of refraction for producing the aforesaid deflection, and it is therefore obvious that manufacturers will be privileged to adopt any correlative proportion of angle to index which will satisfy the demands for any tangent deflection which it may be determined to adopt as a unit.

Supposing the chosen unit of deflection, tang γ, to be slightly greater than the above, say exactly equal to 0.01, or one centimeter, our series of prisms would then be:

1 Dioptry producing a tangent deflection $= 1^{cm}$ in the meter-plane.
2 Diop. " " " $= 2^{cm}$ " "
3 Diop. " " " $= 3^{cm}$ " "

A system of numbering prisms in terms of dioptries (to be designated as PRISM-DIOPTRIES, P-D.) could therefore be adopted which would satisfy all the conditions here set forth.

Such prisms could be measured by noting the deflection they produce upon the index line of a coarse centimeter scale, placed at right angles to the line of sight, at the distance of one meter. (See Fig. 3.)

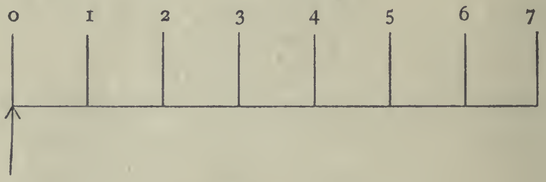


FIG. 3.

If the scale were rendered adjustable, relatively to a vernier, it would be possible to determine fractional parts of the unit deflection.

While a restriction of this character offers the advantage of a ready ocular means of verifying the correctness of the prisms, there are, however, many difficulties to be overcome in manufacturing them. Calculation would disclose the fact that such prisms would require to be ground to degrees, minutes, and seconds, so that comparatively few prisms, out of a lot, at the close of our effort to produce them, would be found to fully meet the aforesaid requirements.

This would so heighten their cost as to render them impracticable, *except as diagnostic instruments in the consultation-room.*

Even these, however, could be substituted by the Prism Mobile, which consists of two prisms rotating before each other in opposite directions, and which will afford the most ready means of filling a demand for definite deflections, inasmuch as the rotation of the prisms, from 0° to 180°, produces all possible deflections from one millimeter upward.

The instrument could easily be graduated to read to centimeters, and tenths, or millimeters of deflection. For the determination of muscular insufficiencies of comparatively low degree, and to render the instrument as light as possible, a special cell, to contain weak rotating prisms, could be devised, similar to that of Dr. Risley, to fit the trial frame.

I may venture to assert that the prism, although the simplest element in Dioptrics, is the most difficult to manufacture, when required to be *exact;* and we shall therefore be obliged, for the present at least, to use existing commercial prisms for spectacle glasses.

I shall subsequently show that these may be profitably utilized, by assigning the unavoidable variations of deflection, consequent to the manufacture of such prisms, to their proper places, as members of the new system.

By actual experiment I have found prisms, represented as being of one degree (1°), to produce deflections varying between 9 and 12 millimeters, and which, if reduced to the basis of our standard of one centimeter, are to be designated as 0.9 and 1.2 dioptries, respectively.

Similar discrepancies of deflection are found to exist throughout the entire series of prisms now in use, so that we shall have adequate variety, covering almost every required interval of the new system; whereas, by the present method, an oculist's prescription, although based upon scientific principles, is constantly exposed to the danger of being reduced to little else than a ticket of chance in an optical lottery.

Farther on an instrument will be described which I have devised to determine the power of prisms in dioptries and fractions, thus making it possible for the optician to select from his stock the one that shall fill the requirements of deflection sought. Indeed, by its use, we may hope to have manufacturers ultimately furnish us prisms, in packages, assorted and marked with the number indicating their power in dioptries.

A Reduction of the Meter-Angle to Dioptries of Prismatic Refraction (*Prism-Dioptries*).

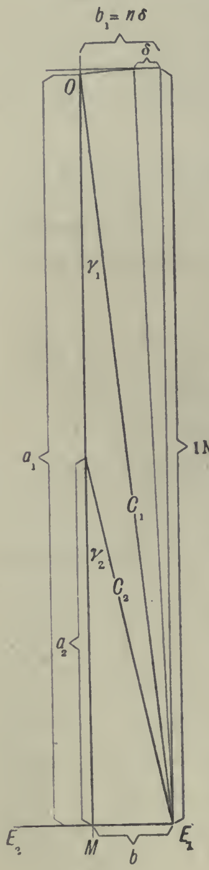


FIG. 4.

In the accompanying figure, 4, E_1 and E_2 represent the centres of rotation of the eyes, and OM the median line, bisecting the base-line E_1E_2 at M.

For the point of fixation O, corresponding to the angle of convergence, γ_1 for the eye E_1, we have

$$sin\ \gamma_1 = \frac{b}{OE_1} = \frac{b}{C_1}$$

or if $C_1 = 1$ meter, $sin\ \gamma_1 = b =$ the deviation required of the eye which is optically adapted for the point O.

The base-line, b, having a constant value for each pupillary distance, 2 b, we have the angle γ_1 solely dependent upon the varying metrical values of C_1, so that the unit-angle of convergence has been designated, by Nagel, the meter-angle when $C_1 = 1$ meter. Hence $1\ ma = arc\ sin\ \frac{b}{C_1} = arc\ sin\ {}_1$; $2\ ma = arc\ sin\ \frac{b}{C_2} = arc\ sin\ \frac{b}{\frac{1}{2}C_1}$ a. s. f.

We may now proceed to find the value of the prism in dioptries, which, being placed before the eye, with its base in, shall produce a convergence to the same point.

The deviation produced by a one-dioptry prism, at one meter, being equal to δ, it is evident that the prism which shall be required to produce a deviation $b = b_1 = \eta\delta$, will have to be η times greater than one dioptry, consequently $= \eta D \triangle$.

If the same deviation is to be produced at the distance $a_1 = \frac{1}{x}$ of one meter, when $C_1 = 1$ meter, for the meter-angle, the prism will require to be X times as great, consequently

$$1\ ma = X \eta \, \mathrm{D}\triangle$$

$$\text{But } X = \frac{1}{a_1} \text{ and } \eta = \frac{b}{\delta}$$

$$\therefore 1\ ma = \frac{1}{a_1} \cdot \frac{b}{\delta} \cdot \mathrm{D}\triangle$$

$$\therefore 1\ ma = \frac{\text{tang}\ \gamma_1}{\delta} . \mathrm{D}\triangle$$

Similarly, if the deviation b is to be produced at the distance $a_2 = \frac{1}{z}$ of one meter, when $C_2 = \frac{1}{2}$ meter, for two meter angles, the prism will require to be Z times greater than η . 1 D△, or $Z \eta$ D△.

Consequently
$$2\ ma = Z \eta \, \mathrm{D}\triangle$$

$$\text{But } Z = \frac{1}{a_2} \text{ and } \eta = \frac{b}{\delta}$$

$$\therefore 2\ ma = \frac{1}{a_2} \cdot \frac{b}{\delta} \cdot \mathrm{D}\triangle$$

$$\therefore 2\ ma = \frac{\text{tang}\ \gamma_2}{\delta} \cdot \mathrm{D}\triangle$$

$$\therefore Xma = \frac{1}{a_x} \frac{b}{\delta} \mathrm{D}\triangle = \frac{\text{tang}\ \gamma_x}{\delta} \mathrm{D}\triangle \ \ldots\ldots\ \mathrm{I}$$

When convergence is confined to comparatively small angles, we may regard the sine, tangent, and angle as being equal to one another. In other words, we may consider $a_1 = C_1 = 1, \quad a_2 = C \ = \frac{1}{2}$, etc, so that

$$1\ ma = \frac{1}{a_1} \cdot \frac{b}{\delta} \cdot \mathrm{D}\triangle = \frac{b}{\delta} \cdot \mathrm{D}\triangle$$

$$2\ ma = \frac{1}{a_2} \cdot \frac{b}{\delta} \cdot \mathrm{D}\triangle = 2 \frac{b}{\delta} \cdot \mathrm{D}\triangle$$

$$Xma = \frac{1}{a_x} \cdot \frac{b}{\delta} \cdot \mathrm{D}\triangle = X \frac{b}{\delta} \cdot \mathrm{D}\triangle \ \ldots\ldots\ldots\ldots\ \mathrm{II}$$

Under these circumstances one dioptry of prismatic refraction differs from the meter-angle by the co-efficient $\frac{b}{\delta}$. This is due to the selection of a comparatively small unit deflection δ. If we had chosen a greater unit deflection, say $\delta = b$, then one meter-angle would correspond to one dioptry exactly. A prism, producing so great a deflection as half the pupillary distance, would, however, give too great an angle for the unit and lowest degree of prism, unless other, as fractions of the unit, were included in the series.

For instance, if $\delta = b$ equal the deflection for one dioptry, prisms of lesser refraction might be designated as 0.25 D., 0.5 D., 0.75 D., when their respective deflections are $\frac{1}{4}\, b$, $\frac{1}{2}\, b$, and $\frac{3}{4}\, b$.

Such a selection would, however, possess no particular advantages, since *b* will generally be a variable quantity for different individuals; besides, it will not be admissible to approximate the factor $\frac{1}{a_x}$ for considerable degrees of convergence.

A strict consideration of the co-efficient $\frac{b}{a_x\,\delta}$, under such circumstances, will be imperative, without regard to any particular choice of the unit deflection, so that for considerable degrees of convergence the prism-dioptries will have to be determined by the formula I.

The following table exhibits the errors committed in estimating the value of the angle of convergence, when formula II. is substituted for formula I., in reducing meter-angles to prism-dioptries.

METER-ANGLES REDUCED TO PRISM-DIOPTRIES, FOR A PUPILLARY DISTANCE OF 64 MILLIMETERS $(b = 32\ {}^{m}/_{m})$
STANDARD UNIT DEFLECTION FOR 1 DIOPTRY $= \delta = 0.01 = 1^{cm}$, AT THE METER-PLANE.

Distance from the Object of Fixation to the Eye.		Sine of the Meter-Angle.	Tangent of the Meter-Angle.	Value of the Angle of Convergence.			Value of the Angle of Convergence when sine, tangent, and angle are accepted as equal.			
In Meters.	In Millimeters.			In Meter-Angles.	In Dioptries.	In Degrees exactly = Δ Deviation.	In Meter-Angles.	In Degrees arc sin.	In Dioptries.	Δ Deviation arc tang.
1	1,000	0.032	0.0320163	1	3.20163	1° 50′ 1″5	1	1° 50′ 1″5	3.2	1° 49′ 58″2
$\frac{1}{2}$	500	0.064	0.0641309	2	6.41309	3° 40′ 9″9	2	3° 40′ 3″	6.4	3° 39′ 43″
$\frac{1}{3}$	333.3	0.096	0.0964465	3	9.64465	5° 30′ 32″1	3	5° 30′ 4″5	9.6	5° 29′ 0″9
$\frac{1}{4}$	250	0.128	0.129061	4	12.9061	7° 21′ 14″4	4	7° 20′ 6″	12.8	7° 17′ 38″9
$\frac{1}{5}$	200	0.160	0.162080	5	16.2080	9° 12′ 24″6	5	9° 10′ 7″5	16.	9° 5′ 24″8
$\frac{1}{10}$	100	0.32	0.337755	10	33.7755	18° 39′ 46″7	10	18° 20′ 15″	32	17° 44′ 40″
$\frac{1}{20}$	50	0.64	0.832919	20	83.2919	39° 47′ 30″	20	36° 40′ 30″	64	32° 37′ 8″5

It is apparent that such a substitution will be admissible, up to five meter-angles, where the difference between the deviation produced by the prism, and the value in degrees of five meter-angles amounts only to 4′ 42″ 7.

When we consider that a muscular insufficiency of five meter-angles is entirely beyond the limits of optical correction, the latter in fact being confined to deficiencies of about one meter-angle, or less, it is obvious that a substitution of the sine for the tangent will be justifiable.

Such being the case, the subject of prismatic corrections becomes wonderfully simple. For instance, for a pupillary distance of 60 millimeters = 6 *cm*, the base-line will be 3 *cm*, when, according to Formula II.,

$$1\ ma = \frac{3\ cm}{1\ cm} = 3 \text{ prism-dioptries.}$$

Similarly, for a pupillary distance of 50 millimeters, the base-line being 25 millimeters, equal to 2.5 centimeters, the meter-angle will be equal to 2.5 P-D.

Thus, for each pupillary distance, we find a different prism necessary to supplant the meter-angle. This is but natural, since greater demands for convergence will be necessary in wide than in narrow pupillary distances.

This leads us to the final and simple rule :

Read the patient's pupillary distance in centimeters, when half of it will indicate the prism-dioptries required to substitute one meter-angle for each eye.

One could scarcely hope for a more convenient method than to find the prism-dioptries, corresponding to one meter-angle, expressed in the patient's features.

There will, however, frequently be occasion to supply less than one meter-angle, as indicated in the following tabulated examples.

Pupillary distance,	$2b$ =	56	60	64	68	millimeters
Base-line	b =	2.8	3.	3.2	3.4	centimeters
1 meter-angle	=	2.8	3.	3.2	3.4	prism-dioptries
$\frac{1}{3}$ " "	=	0.93	1.	1.06	1.13	"
$\frac{1}{2}$ " "	=	1.4	1.5	1.6	1.7	"

According to our standard, a 0.9 D. prism will produce a tangent deflection of 0.9 of a centimeter, or 9 millimeters, and a prism of 1.1 D. a deflection equal to 1.1 centimeters or 11 millimeters. It will therefore be possible to select these, by aid of an adequate instrument, from a paper of our 1° prisms, since the latter are found to produce deflections varying between the same limits.

Prisms of 1.3 to 1.6 dioptries will similarly be found among our 1°½ prisms, and so on.

The Prismometer.

The instrument here described, which we have called a prismometer, to distinquish it from optical theodolites and goniometers of the physical laboratory, affords, we believe, a superiority over the present methods of measuring prisms, inasmuch as it makes it possible to measure prisms up to twenty dioptries at least, to the nicety of fractions.

In the construction of such an instrument there are two essential elements: the one, a meter scale, *AB*, arranged to read from the refracting edge *A* of the prism; the other, the accurate demarkation of the standard unit deflection upon a plane, *P*, which must be movable upon the scale and perpendicular to it.

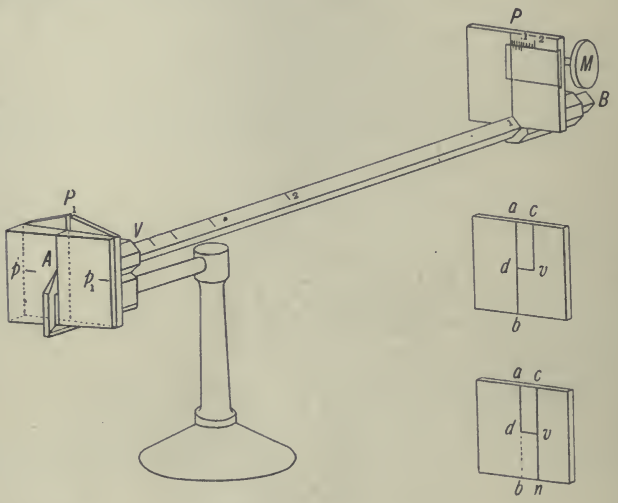


FIG. 5.

FIG. 6.

FIG. 7.

The unit deflection, *dv*, engraved upon the plane *P*, is shown in Fig. 6, as defining the distance between *ab* and *cv*.

For convenience of adjustment and the support of the prism which is to be measured, an upright plate P_1, provided with a sighting slit, is rendered adjustable at the zero end of the scale, so as to admit of prisms of varying thickness to be clamped between it and the zero-edge *A*, and in such a manner that the non-refracting surface of the prism shall rest upon the plate P_1, while its refracting edge is held in contact with the zero-edge, through the resistance of the friction or spring-slide *V*.

Prior to clamping the prism, the true position of its passive or non-refracting plane is to be determined by such rotary adjustment of it, before the eye, until a line, situated at a convenient distance, is sighted as an unbroken one; being precisely the same method which we employ in determining the axes of cylinders. For convenience of registration, ink dots, in collimation with said line, may be applied to the prism.

The prism thus marked is then adjusted with the dots placed upon the horizontal line pp_1, its apex to the right, its upper edge being sufficiently depressed to permit an unintercepted view, through the sighting slit, of the line *ad*, when the prism is to be firmly clamped.

The observer's eye being placed directly before the instrument, in such a manner that the upper edge bisects its pupil horizontally, the lines *ad* and *cv* will be observed in their true positions above, whereas the line *db* will be deflected to the right by the prism below.

This position of the observer being carefully maintained, it is but necessary to advance the plane *P* along the bar *AB* toward the eye, until *cv* and *db* present the unbroken line *cvn* to view (see Fig. 7).

The power of the prism in dioptries, and fractions thereof, may then be read from the scale on the bar *AB*.

In Fig. 5, a modification of the plate *P* is shown with the index of deflection rendered adjustable to a scale with vernier by means of a thumb-screw *M*, which facilitates measurements up to two dioptries. Should the prism to be measured exceed this amount, the index-plate *P* is to be advanced along the bar, say to two dioptries. Our reading of the index *at this distance* being 1.4, for instance, we have a prism which produces a deflection twice as great *at the meter-plane*, or 2.8 P-D.

This arrangement obviates the necessity of graduating the bar closer than single dioptries up to 10 P-D.

To render the instrument more compact it has been found convenient to reduce the size to one half the dimensions set forth.

The general principle evolved affords a new means of verifying the correctness of prisms in a simple manner, and must assuredly serve its purpose, which ever standard unit deflection, at one meter distance, it may be officially determined to adopt, although it is believed that the centimeter has been shown to possess such decided advantages as to be worthy of favorable consideration.

DESCRIPTION OF JAVAL'S NEW OPHTHALMOMETER—MODEL OF 1889.

By H. DRAPER SPEAKMAN, M.D., Ph.D., New York.

ASSISTANT SURGEON, MANHATTAN EYE AND EAR HOSPITAL; INSTRUCTOR IN DISEASES OF EYE AND EAR IN POST-GRADUATE MEDICAL SCHOOL.

THE latest model of Javal's ophthalmometer differs in so many respects from the preceding forms of the instrument, and as it undoubtedly furnishes us with the best objective test of astigmatism that we possess, and is destined to a great future in the hands of oculists, perhaps a description of it may be of interest.

The new model differs from the preceding ones as follows: The barrel of the instrument is much shorter and has a larger diameter; its advantages over the old one are that it gives a much better illumination and is achromatic. This latter result has been obtained with the greatest difficulty. Its objective, composed of a Wollaston bi-axial prism (which doubles the corneal reflections) placed between two bi-convex lenses, has in addition a third bi-convex lens, which shortens the posterior foci of the two images, which foci are marked by a thread of spider's web. The ocular is a compound microscope, and should be accurately focussed on this thread by each observer. There is an arm or arc carrying the measures, which arc is marked in dioptries only. The barrel, instead of being fixed on a horizontal plane, is bent at an angle upward; this is also for better illumination. The large white wheel surrounding this instrument is not found in the old model. This wheel has the degrees marked on its circumference in corresponding quadrants, and has been revolved on its axis until 90° takes the position of 180°; this is because it is to be read for a concave cylinder used in correction. In the corneal reflections the inverted numbers assume their proper position and yield a complete circle of degrees; any and all axes therefore can be easily read directly from the cornea. The outer white circle carrying the numbered meridians is brilliantly reflected on the cornea. The inner black perforated circles absorb light, and are not reflected upon the cornea, while all the white enamelled parts are clearly visible. From the centre to the periphery of the wheel is 45°, and each 5° from the centre to that point is marked on the radii; it is also marked in dioptries. By requiring the patient to look at any designated degree on the radius, the cornea can be measured at that angle. There is also a scale on one of the radii adapting the instrument to pupillometry. There are also two scales on the inner circles marking the 360° of the circle. The outer scale marks every fifth degree, and the inner one every tenth degree. There is in addition a movable circular piece of black metal, which may be attached either behind or in front, and protects the observer from the glare consequent on facing the light, for this instrument requires a strong light, whether natural or artificial. Attached to the centre of the arc and at right angles to it, is an indicator, which moves with the arc and points out the numbers of the meridian on the periphery of the wheel, at right angles to the one indicated by the pointers on the measures. Of the two measures, the one bearing the white rectilinear figure, is fixed in position, and the other one slides back and forth on the arc. This movable one has white enamelled blocks, each one of which is calculated to express one dioptry of corneal curvature, or a power equal to a cylinder of the strength of one dioptry. It is so arranged that it can measure thirteen dioptries of astigmatism. Black velvet has been used as a background for these measuring blocks, on account of its light-absorbing properties. As a practical instrument, this model is far superior to any of the preceding ones, and will give remarkably accurate results to those who master the proper method of using it.

A CASE OF TUBERCULOSIS OF THE CONJUNCTIVA, WITH REMARKS.

By H. KNAPP, M.D.

(*With three figures on plate in the text.*)

TUBERCULOSIS of the conjunctiva, though probably not a very rare disease, has yet so seldom been described that the communication of well-authenticated cases seems not only justifiable but desirable. Though I have read many publications on tuberculosis of the eye, and regarded a number of cases seen in my own practice and that of other oculists as being probably tuberculous, the following is, in my personal experience, the only one in which the nature of the disease was demonstrated with the accuracy and completeness required by modern histology and bacteriology.

John B., æt. nineteen years, consulted me September 2, 1889, on account of a chronic eye trouble.

History. No tuberculous affections in family. He had typhoid-fever and children's diseases when young. For years he had a sore nose and frequent colds in the head. The alæ nasi were swollen and ulcerous. They were permanently cured four years ago by "cutting and cauterization." He has been hoarse for three or four years, less in summer and during the day than in winter and during the evening.

For three or four years his left eye has been more or less red. All that time there was considerable lachrymation and moderate mucous discharge. The lids have been heavy, their nasal edges ulcerous. They have been treated for trachoma with sulphate of copper and other remedies, at times cauterized—all without any benefit.

When I saw the patient the first time he appeared to be a healthy young man. He did not cough, but his voice was somewhat rough, owing, as Dr. M. Toeplitz ascertained later, by congestion, swelling, and small ulcers of the mucous membrane of the larynx. The walls of the nasal orifices, particularly the septum nasi, were contracted by white cicatrices. The nasal passages were free ; the pharynx showed no abnormity.

The right eye was healthy ; the left watered and secreted some mucus. The cornea and ocular conjunctiva were healthy. The inner portion of the edge of both lids, from the commissure to 2 or 3 *mm* beyond the punctum lacrymale, was uneven and pale, presenting small rounded elevations and ulcerous depressions, which were partly free, partly covered with scabs. The mucous membrane of both lids, especially the fornix portion, was dark-red, somewhat swollen and uneven, here and there beset with small, grayish-red nodules, resembling trachoma granules. What attracted the greatest attention, however, were two ulcers in the mucous membrane of the upper lid, one near the free edge of the tarsal conjunctiva, about 5 *mm* in diameter, the other in the retrotarsal fold, about 7 *mm* in diameter. The former was raised over the surface, with slightly projecting grayish nodules, showing decay on the crests of some of them, as well as in the tissue between them. The latter ulcer was deeper than the former, exhibiting extensive necrobiosis in the centre, whereas its edge was uneven, nodular, and slightly raised.

I thought that I had to deal with tuberculosis of the conjunctiva, but favorably considered the diagnosis of Dr. Lustgarten, who happened to be present when I demonstrated the patient at my clinic, and who thought it might be lupus. He based his opinion on the scar at the orifice of the nose. To recognize the real nature of the affection, I wanted to investigate the case in all its aspects, and have done it to the best of my ability.

Without losing time by therapeutic experiments, I cut out the whole tissue surrounding the large ulcer, bottom and wall. I inoculated a particle of the substance into a rabbit's eye. No result was reached, as the rabbit died in a week. Microscopic specimens from the surface secretion, the tissue juice, and the tissue after hardening, made by Dr. J. E. Weeks, showed, on the most careful search, only two or three tubercle bacilli, but they were, in stain and shape, unmistakable.

The excision of the large ulcer produced no reaction. The ground soon produced granulation tissue, which I cut off. The wound soon cicatrized.

Two weeks after the operation, I removed, clean and deep, the swollen parts of the inner edge of both lids, and exsected the basis and walls of the other ulcer, the one near the free edge of the lid. When the bleeding had subsided, I burned the entire surface of the wound which had been made by the extirpation of the tarsal ulcer, and destroyed, with the galvano-cautery, the visible nodules scattered over the conjunctiva. I left untouched the wounds along the edges of the lids. There was no reaction from these procedures. In the course of some weeks the wounds healed completely, leaving no deformity. In particular I may mention that, in spite of the removal of a considerable quantity of tissue, there is no gap, and scarcely any visible defect, at the inner canthus. There is at present, three months and a half after the operation, no perceptible relapse, and the eye feels comfortable. I ordered the patient to brush a $\frac{1}{5000}$ solution of corrosive sublimate over the inner surface of both lids two or three times daily. I think that the conjunctival affection may now be kept in check and even permanently cured by destroying with the galvano-cautery each nodule or little ulcer that may yet appear.

With regard to the **examination of the specimen last exsected,** I have been particularly careful, for I thought that the chances of finding bacilli were more favorable in this than in the previous specimen, the morbid process evidently being of newer date, and the number and vitality of the bacilli greater. The microscopic examination—in which I have been greatly helped by Dr. Weeks, assistant surgeon to the hospital and curator of the museum, as well as the inoculation of animals, demonstrated the tubercular nature of the disease beyond a doubt.

In the surface secretion and fresh tissue juice, treated according to Koch-Ehrlich, a moderate number of bacilli was found. The tissue was laid in Müller's fluid a few days ; then washed in diluted, hardened in absolute, alcohol ; stained with eosine and hemotoxyline for histological and, according to Koch-Ehrlich, for bacteriological purposes.

Transverse sections showed that the piece removed exceeded the morbid infiltration and extended into the healthy tarsus. The superficial layers were densely packed

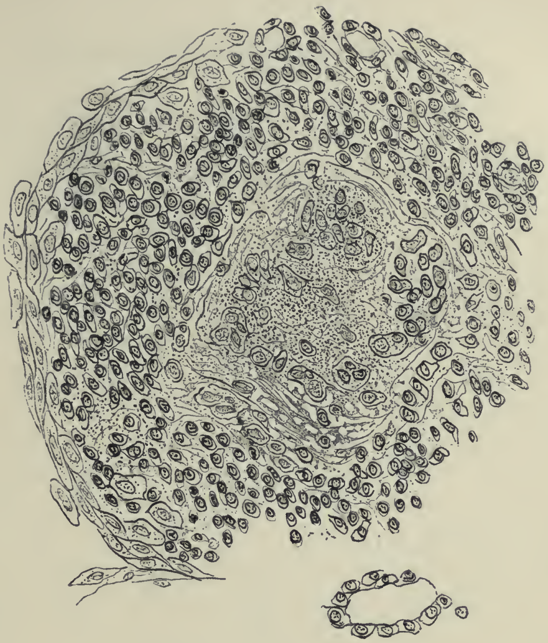


FIG. 1.

From wall of tuberculous ulcer. Accumulation of lymphoid cells, containing patches of granular detritus and gaint cells.

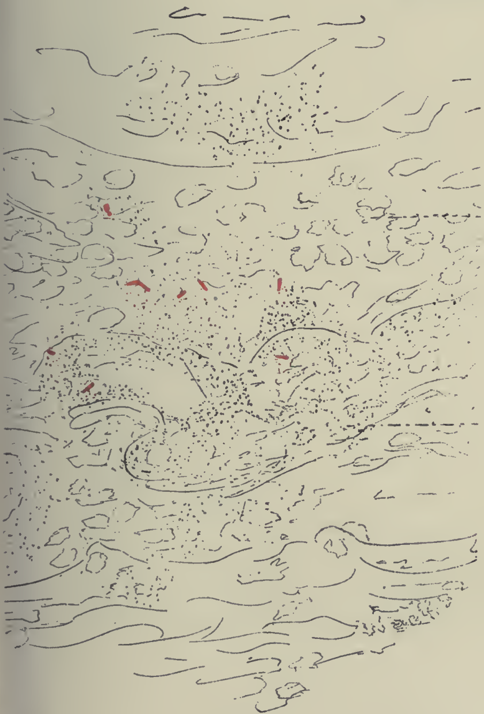


FIG. 2.

Tubercle bacilli in tissue from lid (tarsal conjunctiva.)

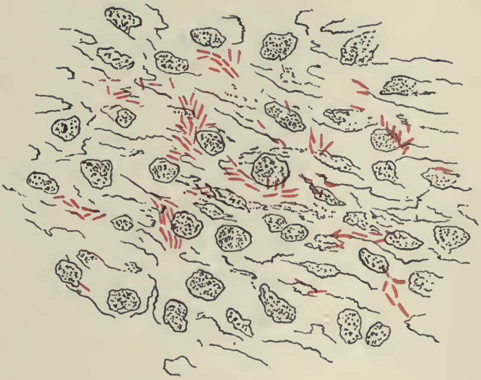


FIG. 3.

Cheesy material from anterior chamber of rabbit's eye.

with lymphoid cells, which, in clusters and bands, penetrated into the healthy tissue as far as the neighborhood of the Meibomian glands, but nowhere between the acini. In many places the lymphoid cells were accumulated in the shape of round patches—nodules, small tubercles—some covered with epithelium (Fig. 1 of accompanying plate. Drawn by Dr. Weeks), others free with a ragged border—ulceration on the surface. The intercellular substance was exceedingly scant, more homogeneous than dotted or striated. The centre of some of the round patches showed granular detritus, in which multi-nucleated (giant-) cells were noticed (Fig. 1).

In the slides which were prepared according to Koch-Ehrlich, most of the bacilli were strewed singly over the field (Fig. 2). Yet in some places, in the giant-cells, they lay together in clusters of from three to six and more.

Inoculation. — Immediately after the removal of the smaller ulcer, I introduced small tissue particles from it into the eyes of two healthy, middle-sized rabbits. With a cataract knife I made a small incision into the periphery of the anterior chamber, introduced the small piece of tissue through the wound with a delicate pair of microscope forceps, and pushed it over the iris to some distance from the wound, or into the pupil. This procedure requires some delicacy and patience, as the intraocular pressure tends to reject the particle and produce prolapse of the iris. The wound closed nicely, the episcleral injection disappeared in a few days, and for four weeks the eyes were free from irritation, the animals lively, the implanted pieces became smaller, and in two eyes they could not be seen any more. About four weeks after the inoculation there appeared in three eyes marked congestion, and in the course of a week a great number of yellowish miliary nodules all around in the iris. Then the eyeballs swelled, the corneæ became opaque and vascular. In three weeks the corneæ bulged at the seat of the wound, burst, and cheesy material covered this part of the cornea like a mushroom. In the eighth week after the inoculation, I removed one eye and found numerous small yellow tubercles in the iris, the retina detached, and the ciliary processes partially covered with a grayish opacity. The cheesy secretion in the anterior chamber and upon the cornea contained multitudes of bacilli (Fig. 3); also the nodules in the iris contained a goodly number. In these nodules there was the same dense accumulation of lymphoid cells as in the original ones of the human tarsal conjunctiva. They contained no nests of cheesy detritus, and no giant-cells. The animal from which one eye had been removed had lost flesh, improved in general condition after the enucleation, but died two weeks later. All the organs were found healthy except the lungs, which showed a limited number of glassy miliary tubercles in the visceral pleura. The other rabbit is still living. In one eye the cornea is destroyed, and white cheesy masses emerge from the interior. The other eye is unaffected, and the animal lively. The early death of the other rabbit may have been the consequence of the binocular blindness interfering with nutrition and motion.

I beg leave to add some **remarks** to this case:

1. The **question, whether tuberculosis** or **lupus,** is **immaterial.** The same microbe is the cause of both diseases, and lupus is considered identical with chronic tuberculosis. Those cases have been chiefly classified as lupus of the conjunctiva in which the affection proceeded from the external side of the lid to the internal. In our case it is possible that the infective material was transmitted from the meatus of the nose through the naso-lachrymal canal into the conjunctival sac. This way of propagation is made probable by the development of tubercular ulcers around both puncta lacrymalia.

2. Our case **cannot be considered as one of primary tuberculosis,** for symptoms of nasal and laryngeal tuberculosis were noticed long before the manifestation of the affection in the conjunctiva. Primary tuberculosis of the conjunctiva seems to be very rare. Caesar Amiet[1] mentions among forty-seven cases of conjunctival tuberculosis only two primary ones, of which one—the case of Haab[2]—must be

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[1] "Die Tuberculose der Bindehaut des Auges," Inaugural Dissertation, Solothurn, 1887.

[2] *v. Graefe's Arch.*, xxv., 4, p. 224.

deducted, for the primary seat of the affection was in the choroid.

Prof. E. Fuchs, however, stated at the meeting of the Heidelberg Ophthalmological Society in 1887,[1] that in Vienna he sees a comparatively great number of conjunctival tuberculosis, and that in the majority of his cases no tuberculosis can be discovered in other organs of the body. He thinks that particles of dust, loaded with tubercle bacilli, enter the upper conjunctival sac, locate, as foreign bodies mostly do, in the shallow furrow of the upper tarsal conjunctiva, along and near the free edge of the lid, and inoculate the conjunctiva by mechanically detaching a small portion of the conjunctival epithelium. At the same meeting Valude had communicated to the society his experiments of inoculating the conjunctiva with tubercle bacilli. They had been successful only when the material was introduced under the mucous membrane. I am afraid that a subconjunctival squint operation has led to the same result in a case of mine which is still under observation, and on which I shall have something to say at a later period.

3. On the **differential diagnosis between tuberculosis and trachoma of the conjunctiva,** a very instructive paper was published in *v. Graefe's Archives,*[2] November, 1888, by K. Rhein, on "Follicular Disease of the Conjunctiva." He there publishes two cases of tuberculosis, and says: "The follicular formations in tuberculosis of the conjunctiva can, by structure and behavior, neither macroscopically nor microscopically, be distinguished from the so-called trachoma follicles. The differential diagnosis of both diseases can, in the stage of the formation of follicles, only be ascertained by the discovery of the micro-organism which causes the affection, eventually only by the experiment on animals" (*l. c.*, p. 90). I may add, if I am correct in the interpretation of the above case of strabismus, that the tubercle granules lie as well circumscribed, jelly-like, very transparent nodules in the conjunctiva, and are not influenced by daily touchings with the sulphate of copper crystal, whereas the recent cases of trachoma are rapidly cured by this treatment.

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[1] Sitzungsberichte, 1887, pp. 68 and 69.

[2] Vol. xxxiv., part 3, p. 65.

4. The **prognosis** will chiefly depend on the question whether the conjunctival tuberculosis is a primary and localized affection, or only a part of a general disease. Cases of cured tuberculosis are mentioned in literature, for instance, by E. Fuchs in the above-cited communication (*l. c.*, p. 69). Yet the frequency of relapses is not overlooked by authors. General tuberculosis may follow when the local disease is not rooted out. Whether or not a spontaneous recovery may occur, can neither be asserted nor denied, for the disease has not yet been recognized and observed long enough to warrant any statement on this point. That tuberculosis in other organs can be recovered from, seems to-day generally admitted by the profession. Apart from the prognosis quoad vitam, we possess experience enough to say that tuberculosis of the conjunctiva not infrequently damages seriously the visual organ by partial destruction of the lids and by affections of the cornea.

5. The **treatment** should consist, **prophylactically,** in the undelayed destruction of all kinds of foci of local tuberculosis, *e. g.*, lupus nodules and ulcers, caries and necrosis of the orbital wall, the tear sac and nasal passages, scrofulous glands, and the like; **curatively,** in the destruction of the local products—the nodules, ulcers, and infiltrations—by means of the knife, the sharp spoon, and caustic—lunar or actual. The galvano-cautery in this respect is handiest and most efficient. In the after treatment, antiseptics, washes of bichloride of hydrargyrum 1:5,000, and iodoform powder, to be dusted on the affected conjunctiva, have been used and praised.

The **literature** on conjunctival tuberculosis is still scant and easily accessible. Among the recent publications I desire to draw attention to the following papers:

1. H. PAGENSTECHER and AUG. PFEIFFER, "Lupus oder Tuberculose."—*Berl. klin. Mon.*, 7 May, 1883. The tubercle-bacillus was not found in the conjunctiva, but in the iris of rabbits after inoculation.

2. GAYET, "De la Tuberculose conjunctivale."—*Bull. and Mem. de la Soc. Franç. d'Ophthalm.*, 1885. Contains the history of tuberculosis of the conjunctiva, the detailed de-

scription and an excellent colored drawing of a case, and critical remarks.

3. REUSS. *Wiener Med. Blätter*, Bd. x., 1887.

4. A. WAGENMANN, "Contributions to the Knowledge of the Tubercular Affections of the Visual Organ," in *v. Graefe's Arch.*, xxxiv., 4. p. 145, 1888, which contains three cases of tuberculosis of the conjunctiva from LEBER'S clinic at Göttingen.

5. HILL-GRIFFITH, "Case of Primary Lupus of the Ocular Conjunctiva." With literary references on lupus and tuberculosis of the conjunctiva.—*Medical Chronicle*, Nov., 1889.

Modern text-books mention the disease, and some of them, for instance that of Fuchs, describe it well. As we now have the means of diagnosticating it, we shall soon devote to it the attention it deserves.

A CASE OF ALEXIA (DYSANAGNOSIA).

By Dr. SWAN M. BURNETT, Washington, D. C.

On the morning of the 23d of August, 1888, the Rev. W. A. H. aged eighty-two, came to me with a request from Dr. Busey that I examine into the condition of his eyes. His vision, it was stated, had not been satisfactory since an attack for which the doctor had attended him some weeks before. He was rather feeble-looking, though not more so than would be expected in a man of that age, but he had come to my office alone from a distant part of the city, and had had no unusual difficulty in finding my residence, the number of which was written by Dr. B. on a paper, which he held in his hand at the time he presented himself. The statement he made then was that he had not been able to read since the time mentioned, and that he feared "the nerve was injured." The history of the attack is as follows: He had gone out into his front-yard, some weeks before, to assist his servant in watering the grass. He staggered, but did not fall, and, feeling uncomfortable, went into the house. He passed a rather comfortable night, and came down stairs the next morning as usual, read the service, and had the customary family devotions. Soon afterwards, however, he complained of feeling badly, was taken up stairs, and almost directly went into strong general convulsions. Of these he had three during the afternoon and evening, and afterwards passed into a condition of stupor from which he could be roused with difficulty and for only a moment. At the end of three days he became conscious, but was very weak. On the fourth day, in the evening, he read some from the prayer-book, but was very much fatigued thereby. On the next morning he attempted to read again, but found he could not.

On examination I found that he failed to name any letter of Snellen's test-type correctly, even the largest. The refracting media were unusually clear for a person of that age, and the fundus of the eye did not present any thing sufficiently abnormal to account for such a marked deterioration of vision as seemed to be present. A trial with lenses failed to make any change in the unsatisfactoriness of his vision. The ophthalmoscope and skiascopy showed a slight myopia, but the ophthalmometer revealed no corneal astigmatism above what might be considered normal.

Having mentioned, during the course of the examination, which was very tedious for a person of his general intelligence, that he thought the difficulty lay in the tranposition of a letter or word from one line to another, I tested him for diplopia, but found none. His visual field was normal. In my despair of finding any satisfactory cause for his trouble, or even the nature of it, I tried his near vision and found it equally bad; but what struck me as being curious was that he made as many mistakes with the large type as with the small. Finally, with somewhat of impatience with myself for my inability to discover the nature of the affection, I asked him to spell some words of No. 14 Jäger. Then it was that it came to me, in a flash, what the real difficulty was. It was not that he could not see the individual letters of the word, or the word itself, but that they failed to convey to him the same ideas they had for the last seventy or seventy-five years. In other words, the fault lay, not in the globe of the eye, but in the brain. I then set about making systematic tests, and with rather curious, not to say startling, results. I found that he was unable to read any thing correctly. A word here and there might be properly called, but the sense of even the shortest sentence would be ridiculously misinterpreted.

For example: The morning paper was lying on the table, and I asked him to read aloud to me the following sentence: "Judge Thurman will formally open his campaign at Port Huron to-day." This he read to me as follows: "John, John then the hatter his hat going to be h–green." No. 12 of Jäger reads thus: "The keys and he began playing a sad and infinitely lovely movement." His interpretation of it was: "Was told to be and haying a a was to be be ing in mo on when he was crydt."

Even these attempts at reading were made slowly and hesitatingly, and often with the necessity of an effort to decipher a word by spelling, just like a child learning to read.

He was himself aware of this being the veriest nonsense, and having been a close student and constant reader all his life, the annoyance had not a little tinge of humiliation added to the unavoidable ludicrousness. I have submitted him to a number of different examinations with a view of finding the exact limit of his defect. His trouble is confined entirely to an inability to interpret the meaning of printed or written words by means of the impressions they make on the retina. When any thing is read to him he understands it perfectly and can repeat it accurately, and his memory of things he has read before is unimpaired, and all other impressions made on his retina are properly interpreted. He can even read numbers correctly, and can tell the amount of a check, though unable to tell to whom it is drawn or by whom. And not only are the Arabic numerals recognized without difficulty, but he is able to interpret correctly the Roman numerals also. All the letters he can distinguish individually with ease except the letter "s." To look at this is always disagreeable. The word "the" is seldom or never misinterpreted. All kinds of pictures he understands and enjoys.

He can write either originally or from dictation, but is as unable to read his own writing as he is that of any one else or printed matter. It is necessary, however, that he write continuously. If interrupted, he cannot go back and begin where he left off. Yet it is possible for him to break a line in writing if he is not interrupted in his own line of thought. He can divide a word properly at the end of a line and start out on the proper letter at the beginning of the next. For instance, he wrote, at my dictation:

"Of man's first disobe-
dience and the
fruit of that forbid-
den tree."

Under my instructions, his wife has been making an effort to reëducate his reading faculty, and for that purpose procured a school primer and began as with a child learning its letters for the first time. He can call the letters of the alphabet correctly separately, and can spell a few words in succession properly, sometimes as many as five or six, but after this the brain becomes fatigued and he shuts his eyes or hands the book back. If he is fatigued in body or mind before the effort is begun he can not make out more than two or three words. It must be said, however, that the improvement under this plan has not been encouraging. His memory for what he has read before is by no means impaired, but in attempting to verify a scriptural quotation, for example, as he did some days ago, he turned to the place where he thought the passage was, in Job, and indicated and read as the passage he was looking for, one which had not the slightest resemblance to the real one, which was in Amos. He was not aware of his mistake until it was pointed out to him by his wife.

These notes were made nearly a year ago, and the patient succumbed to an attack of pneumonia on January 20, 1890. In the meantime, however, no essential change had taken place in his condition. His general mental faculties remained unusually bright and clear to the last, and his bodily vigor was as good as it had been for many years past until seized with the prevailing influenza. His reading faculty, however, was never regained to a degree beyond that reported above.

This case seems to be one of alexia, pure and simple. No other faculty, so far as the closest scrutiny and the most careful examination could determine, was affected except that of reading. This fact would seem to demonstrate quite conclusively the existence of a "reading centre" separate and distinct from any and all other centres. Where that centre is situated, we are, unfortunately, not able to bring any pathological facts from this case to demonstrate. His death occurred whilst I was absent from the city, and a post-mortem examination would, under any circumstance, have been difficult to procure.

The literature on the subject, while not scanty, is yet too deficient in clinical and pathological details to allow of any positive deduction as to the seat of lesion. Several autopsies have been made in cases where alexia was a part of the clinical history, and the changes found will serve at least as indications for search in cases which come under observation in future. In Nieden's[1] case, Broca's third posterior left convolution was uninjured; there were apoplectic spots about the corpus striatum, extending to inferior frontal convolution; the gray matter was not destroyed.

In Berlin's cases, six in all, there were three autopsies.[2] In the first there was atheroma of the left arteria fossæ Sylvii, brain substances unaltered; in the second the whole brain surface was inflamed; in the third the alteration was confined to the left side—fresh hemorrhages and old morbid processes, thinning of roof of left ventricle, and thromboses of left art. front. and communicans post. and general atheroma.

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[1] These ARCHIVES, vol. xvii., p. 307 *et seq.*

[2] Ueber Dyslexia. *Baden-Baden u. med. Corresp. Bl. d. Würtm. ærztl. Landes-Versamml.*, 1883, No. 27.

These data are altogether too indefinite to base any thing like a positive opinion on, but it would seem that we can look for the lesion in cases of alexia, on the left side, and in particular in the region between Broca's frontal convolution, the facial centre and the central convolutions.

Landolt has reported a case in Donders' Jubilee-memorial volume, and there are other cases scattered in various journals of general and neurological medicine, but none that I have been able to refer to where the lesion was so isolated as in the case I have reported.

I agree with Nieden in the opinion that the term "dysanagnosia" is etymologically more correct and scientifically preferable to either "alexia" or "dyslexia," the two latter being hybrids of Latin and Greek, whose adoption in our nomenclature should not be encouraged.

It would also seem desirable to separate clinically the graver from the lighter forms of the lesion. Berlin reports several cases where there was simply a fear or disinclination of reading, though all the words could be properly interpreted. These are certainly not the same in degree as those cases like the one whose history has just been reported, where the ability to distinguish even a few words in succession is absent.

SYSTEMATIC REPORT ON THE PROGRESS OF OPHTHALMOLOGY DURING THE FIRST QUARTER OF THE YEAR 1889.

By H. MAGNUS, Breslau; C. HORSTMANN, and P. SILEX, Berlin;

WITH THE CO-OPERATION OF

S. M. Burnett, Washington; Dantone, Rome; Hirschmann, Charkow; E. Marckwort, Antwerp; P. v. Mittelstädt, Metz; L. Werner, Dublin; C. H. A. Westhoff, Amsterdam; Schjötz and Ole Bull, Christiania, etc.

(*Concluded from p.* 514 *of vol. xviii.*)

Sections XII.—XX. Reviewed by Dr. P. SILEX.

XII.—IRIS.

125. Collins, E. T. Primary sarcoma of the iris. *Royal London Ophth. Hosp. Rep.*, vol. xii., iii., p. 273.

126. Franke. A case of partial irideremia. *Zeh. klin. Monastbl.*, vol. xxvii., p. 93.

127. Hasbrouck, S. A report of cases of persistent pupillary membrane and persistent hyaloid artery. *Jour. Ophth., Otol., and Laryng.*, Jan., 1889.

128. Heddæus. On reflex immobility of the pupil. *Centrabl. f. Nervenhk.*, etc., vol. xii., No. 3.

129. Schmidt–Rimpler. The development of serous cysts of the iris. *v. Graefe's Arch. f. Ophth.*, vol. xxxv., i., p. 145.

130. Theobald, S. A case of double congenital irideremia in a child whose mother exhibited congenital coloboma of each iris. *Trans. Amer. Ophth. Soc.*, 1888.

COLLINS (125) was consulted by a young man, æt. twenty-one, concerning a brown spot on the left iris, which had gradually increased in size since he first noticed it three years before. The iris was greenish, mottled with pigment, and the pupillary margin, below and internally, presented a rounded, brown projection. The eye was normal in every respect including vision. After excision of the eyeball the microscope revealed a round-celled sarcoma of the iris involving the ciliary region. The author reviews the literature of previous cases (17 in number) and sums up as follows: The disease occurs most frequently in females, and is more common between 20 and 40; it is more often melanotic than white; the lower part of the iris is the most frequent position; iridectomy may prove successful in many cases, if the whole growth can be removed. Three illustrations accompany the paper. WERNER.

In FRANKE'S (126) case there was but a narrow strip of iris left above in both eyes, while the lower half was completely absent. $V = \frac{6}{9}$. Every thing else normal.

HASBROUCK (127) reports two cases of persistent pupillary membrane—one in which there were only two or three threads, in the other quite a thick network on the posterior surface of the iris, and not attached to the pupillary edge. The persistent hyaloid artery was attached to the posterior surface of the lens, its "vermicular movements" being very noticeable. BURNETT.

HEDDÆUS (128) discusses with Möbius the conception and nature of reflex immobility of the pupil. Monocular reflex insensibility is opposed to reflex immobility of the pupil. In monocular reflex insensibility, for instance, after embolism of the central retinal artery, the two pupils of equal size react promptly when both eyes are illuminated or shaded, or the healthy eye only. The pupil of the blind eye does not react when light falls upon it, but it responds to an accommodative effort. In monocular reflex immobility the pupils are of unequal width; when one or both eyes are shaded or illuminated, only the healthy eye's pupil reacts, but both respond to convergence. In tabic myosis the pupil could be dilated and made movable with cocaine.

From the observation of a case SCHMIDT-RIMPLER (129) concludes that the spontaneous or idiopathic serous cysts of the iris are collections of lymph in closed crypts of the iris. The endothelium is derived from the endothelium covering the surface of the iris and dipping into the crypts.

The interest in Theobald's (130) case lies in the fact that the colobomata of the iris in the mother were upward ; no other congenital defect. Burnett.

XIII.—CHOROID.

131. Brockman, E. F. A case of ossification of the choroid. *Brit. Med. Jour.*, 1889, p. 585.

132. Browne, E. On the occurrence of central choroiditis with cerebral symptoms in children.

133. Litten. On a case of melano-sarcoma of the liver. *Deutsche med. Wochenschr.*, 1889, No. 3.

134. Ludwig, G. Rupture of the choroid, pseudo-cysticercus in the vitreous, and nystagmus of the right eye. Hippus. *Centralbl. f. prakt. Augenhk.*, vol. xiii., p. 34.

135. Ruiz. Ossification totale de la choroide. *Rec. d'opht.*, Jan., 1889, p. 29.

In Brockman's (131) case, ossification of the choroid occurred in an eye which had suffered from specific irido-choroiditis ; after its removal, the other eye, which was also affected, began to improve, and finally recovered good vision. Werner.

Browne (132) has observed central choroiditis, commencing in the form of white or buff-colored spots in children who were subject to night-terrors, occasional vomiting, and variable condition of bowels. No history or evidence of syphilis. Werner.

In Litten's (133) patient, a sarcoma of the choroid was diagnosticated in 1884, and soon afterwards the eye was enucleated. Although metastasis of the liver was discovered in the same year, death did not ensue until 1888. It is worthy of note that the urine, always acid and clear when discharged, became black after standing a few hours, a condition which disappeared at once when chromic or fuming nitric acid was added. On adding sulphate of copper, the coloring substance of the urine formed a salt resembling that of Trommer's test, as the urine, after being rendered alkaline and after the addition of the sulphate of copper, turned to a bright orange-red color, without, however, the well-known precipitate. The melanuria has not as yet been described by oculists ; it probably does not appear until metastases have developed in the inner organs, especially the liver.

Ludwig (134) reports that the complicated condition he describes developed after a blow on the right eye and sickness extending over three months, coupled with unconciousness (meningitis ?) ; the vertical nystagmus he ascribes to irritation in the corpora quadrigemina due to an inflammatory exudation.

Ruiz (135) observed glaucoma in the right eye of a man aged seventy-three, which resulted sympathetically from the left eye, which had become atrophic after an injury received twenty-five years ago, and was sensitive to pressure. When the stump was removed the whole choroid was found to be ossified, only a small spot in the region of the macula lutea being free. The effect of the enucleation on the right eye was excellent. Marckwort.

XIV.—GLAUCOMA.

136. Dobney, S. G. Acute glaucoma induced by the instillation of atropine, in a man seventy-two years old, whose mother had also had glaucoma. *Amer. Pract. and News*, Feb. 16, 1889.

137. Logetschnikow. The indication for iridectomy and sclerotomy in glaucoma. *Wjestnik Ophth.*, March, April, 1889.

138. Nettleship. On the prognosis in chronic glaucoma (continued). *Royal London Ophth. Hosp. Rep.*, vol. xii., Part 3, p. 215.

139. Picqué, Lucien. Pathogénie du glaucome, étude critique et expérimentale. *Arch. d'opht.*, vol. ix., No. 1, January, February, 1889, p. 21.

140. Theobald, S. Is astigmatism a factor in the causation of glaucoma ? *Trans. Amer. Ophth. Soc.*, 1889.

141. De Wecker. Glaucome et inflammation. *Arch. d'opht.*, vol. ix., No. 2, March, April, p. 170.

In Dobney's (136) case atropine had been used once before for the alleviation of an acute conjunctival catarrh, without harm. In this instance, however, glaucoma developed after twice using a 4-gr. solution of the drug. No treatment was instituted, but antipyrine was administered in 10-gr. doses. Two such doses produced sleep and relief to the glaucomatous symptoms.

Burnett.

Logetschnikow (137) has operated on over 1,000 cases of glaucoma, 284 sclerotomies (257 according to Wecker-Mauthner) being among these operations. He groups his cases of sclerotomies in five tables, an analysis of which, based on his experience, leads him to the following conclusions : Operative treatment is indicated in all forms and stages of glaucoma, except in the initial stage, when myotics are sufficient, sometimes even for years. Operative treatment does not produce a radical cure, but only checks the disease for a longer or shorter space of time. If necessary the operation can be repeated many times with good result. Only in cases of malignant glaucoma, which, however, cannot be diagnosticated before the operation, the latter is directly harmful. In general, iridectomy is the safer, better method. It is preferable, particularly in inflammatory glaucoma, in the chronic as well as in the subacute form. A correct iridectomy is very difficult, sometimes inpossible, in cases of acute glaucoma and in absolute glaucoma with beginning degeneration. Sclerotomy, however, can easily be performed in such cases, and as the statistics show, with much benefit, especially in absolute glaucoma. Sclerotomy performed during an acute attack must only be considered a preliminary operation, which should be followed by an iridectomy for safety's sake. In non-inflammatory glaucoma, sclerotomy is to be preferred (were it only for cosmetic and optical purposes). It is also to be preferred when escape of vitreous may be expected. Iridectomy is necessary in secondary glaucoma following anterior or circular posterior synechiæ or cystoid scar. Of all pericorneal methods, that of Wecker-Mauthner is the best. The proposition of Snellen, to make iridectomy with preservation of the pupillary margin, is worthy of attention, and can be easily combined with double sclerotomy, as Logetschnikow has found. Myotics take the place of an operation in the prodromal stage, are necessary in sclerotomy for the sake of more perfect technique, and are useful in treatment after the operation. HIRSCHMANN.

NETTLESHIP (138) treats of various questions connected with glaucoma in general. Seven cases of hemorrhagic glaucoma are recorded. In fifteen cases of primary glaucoma occurring in persons under thirty years of age nearly two thirds were myopic; four of them were under twenty. Only five of these were seen early enough for treatment to be useful, and all underwent iridectomy with good results, although two were considerably myopic. When glaucoma occurs in myopic eyes it begins very early (in ten among eighteen cases observed by the author). In most of the cases the glaucoma was double and the myopia equal in the two eyes, but there seemed to be no quantitative connection between the two conditions. A case of glaucoma is recorded occurring seventeen years after cataract-extraction, and also a case of undoubted glaucoma commencing with central scotoma. WERNER.

Picqué (139) considers simple glaucoma a chronic affection of the optic nerve, the nature of which is not explained by any of the accepted theories. He thinks this view is supported by the contraction of the visual field indicating a lesion, especially of the temporal fibres, the reverse of the retrobulbar neuritis affecting the fibres supplying the macula. The occurrence of cases marking a transition from typical simple glaucoma to genuine atrophy of the optic nerve also seems to sustain this view. The cause of acute, genuine glaucoma Picqué seeks with Panas in the disturbance of the circulation arising from whatever cause in a territory of atheromatous blood-vessels. The consequence is stagnation and intrachoroidal and subretinal œdema with increase of tension. The affection of the blood-vessels is proven by the hemorrhages in hemorrhagic glaucoma and by the bleeding after iridectomy. Experimentally such hemorrhages never occur in healthy eyes when a high tension is suddenly reduced, and in direct contrast with the glaucomatous appearance in the human eye the blood-vessels were found contracted, even to the disappearance of the arteries and veins. Neither a dilatation of the pupil nor an excavation resulted from increased tension; neither did any glaucomatous hyperæmia of the surface of the globe result. The haziness of the cornea disappeared as soon as the tension was diminished. The anterior chamber did not become shallow, but deeper in consequence of the bulging of the cornea. The author does not consider the increased tension the essential and primary feature in acute glaucoma either. Chronic inflammatory glaucoma he does not consider genuine glaucoma; it is only a complication of simple with acute glaucoma. The symptoms are those of a genuine inflammation, and resemble those of an inflammation of the uveal tract attended with increased tension. He does not express a decided opinion as to whether acute glaucoma is originally of an inflammatory character, or whether it becomes so when it passes into chronic inflammatory glaucoma. He however positively asserts that the obstruction theory is not applicable to any kind of glaucoma. v. Mittelstädt.

Theobald (140) is of the opinion that astigmatism, contrary to the rule, is a possible factor in the causation of glaucoma, and cites several cases in support of his belief. Burnett.

De Wecker (141) attacks the conclusions of Picqué (*v.* above), and considers the so-called inflammatory symptoms of glaucoma only a manifestation of disturbance in the mechanism of nutrition, which have nothing to do with inflammation. The latter, as far as has been determined for the eye, seems due to infection alone. Mechanical influence can certainly be excluded as a cause. Clinical observation on man, for instance, in cases of traumatic dislocation of the lens without external injury, followed by glaucoma, furnishes a less objectionable experiment than those on animals. De Wecker considers glaucoma nothing but a disturbance of equilibrium between secretion and discharge of the ocular liquids.

v. MITTELSTÄDT.

XV.—LENS.

142. BRIBOSIA. Avant, pendant et après l'extraction de la cataracte. *Ann. d'ocul.*, vol. ci., p. 103.

143. MCKEOWN. On extraction of senile cataract without iridectomy, with notes on the result of forty cases. *Brit. Med. Jour.*, Feb. 9, 1889, p. 307.

144. KUNN. Heredity of zonular cataract in one family. *Wiener klin. Wochenschr.*, 1889, No. 3.

145. LEE, CH. Extraction of soft cataract by intracapsular irrigation. *Brit. Med. Jour.*, March, 1889, p. 706.

146. LEREBRIKIKOWA, E. Report on the second hundred of cataract-extractions at the Government Hospital at Perm. *Wjestnik Ophth.*, Jan., Feb., 1889.

147. LITTLE, D. On extraction of senile cataract, with the results of 1,248 operations. *Brit. Med. Jour.*, Feb., 1889, p. 407.

148. MACNAMARA. On extraction of cataract and rheumatic sclero-keratitis. *The Westminister Hosp. Rep.*, vol. iv., p. 27.

149. NOYES, H. D. Considerations concerning extraction of hard cataract, with an analysis of 309 cases. *N. Y. Med. Rec.*, March 30, 1889.

150. POMEROY, O. D. Three consecutive and successful extractions of luxated lenses by means of Agnew's bident, with two additional cases from other sources. *Trans. Amer. Ophth. Soc.*, 1888.

151. SAUNDERS. Traumatic dislocation of lens ; fulminating glaucoma ; removal of lens ; recovery with good vision. *Brit. Med. Jour.*, March, 1889, p. 470.

152. THOMAS, CHAS. M. A report of 120 cases of cataract-extraction. *Jour. Ophth.*, Oct., 1888.

153. Webster. A case of extraction of partially absorbed calcareous lens complicated with posterior synechiæ, with resulting vision of $\frac{20}{20}$. *Trans. Amer. Ophth. Soc.*, 1888.

Bribosia (142) gives his views in general on cataract-operations. For the artificial ripening of cataract, he prefers puncture of the capsule (with the stop-needle) to Förster's method.

Marckwort.

In McKeown's (143) cases the flap was totally corneal, and intraocular irrigation was used. In the forty cases there was no suppuration, one case of iritis, and one of escape of vitreous. Prolapse of iris occurred from accident in two cases, and without known cause in three, but they all recovered good vision. The visual acuteness was considered better than by v. Graefe's method, and patients often select glasses 2 to 4 D lower than after the latter operation. Werner.

Kunn (144) observed zonular cataract in a grandmother, mother, and two sons. The mother and sons had distinct symptoms of rhachitis.

Lee (145) gives results of 21 cases of extraction in which irrigation was used to remove sticky cortical remains which could not readily be removed by the usual proceeding. He considers iridectomy an essential precursor to irrigation. There was no loss of vitreous, and only one case of iritis. Of 21 cases, 10 obtained $V = \frac{20}{30}$; 3, $V = \frac{20}{40}$; 6, $V = \frac{20}{50}$; 1, $V = \frac{20}{70}$; 1, distant V not stated, J 14 (iritis). Werner.

Little (147) performs the 3 *mm* flap with iridectomy. The general results of two series are given : (1) in 1,048 extractions performed in hospital (1873–1887) there was loss of vitreous in 5 % (1 in 5 followed by inflammation), iritis in 11.5 %. Results : V (not less than $\frac{1}{10}$) good in 90 %, moderate (V between 20 Jaeger, and counting fingers) in 4.5 %, doubtful (prospect of doing good by iridectomy) in 2 %, failures (half due to corneal suppuration or suppurative iritis) in 3.5 % ; 4 % obtained $V = \frac{6}{6}$, and 7 % $= \frac{6}{9}$. (2) In 200 private cases there was loss of vitreous in 4 %, iritis in 10 %. Results : good in 94.5 %, moderate in 3.5 %, doubtful in 1 %, failures in 1 % ; 17.5 % obtained $V = \frac{6}{6}$, and 25 % $= \frac{6}{9}$. In two of the successful cases iritis came on, in one twelve months afterwards, and in the other four months later, leading to sympathetic ophthalmia, with loss of both eyes. Since 1887 the author has performed 133 extractions in private and hospital, with antiseptic precautions, without suppuration, and only once did iritis occur. Werner.

In the first part of his paper, MACNAMARA (148) advocates extraction without iridectomy, with antiseptic precautions; he next gives a description of the common form of rheumatic sclerokeratitis. If the tension be increased in these cases, atropine and cocaine should be avoided. WERNER.

NOYES (149) bases his remarks upon the statistics of 309 operations done by eight operators at the New York Eye and Ear Infirmary. Most of them were done by the upward section and with an iridectomy. Most of the operations were done under cocaine, for which he considers 2 % quite strong enough. The eyes were washed with an antiseptic solution, either 4 % boric acid or 1 : 5,000 bichloride or 1 : 20,000 biniodide. The anterior chamber was washed out in some cases. He prefers to make the corneal section embrace about $\frac{2}{5}$ of the circumference. In the 35 simple extractions, there was no failure. Successes 27, medium 7, unknown 1. Of the whole number the successes (V $= \frac{1}{10}$ and better) were 79 %, less than $\frac{1}{10}$ 14 %, failures 7 %. BURNETT.

POMEROY'S (150) cases are related in full, and the success in the use of the bident, described by Dr. Agnew in a previous number of the transactions, is such as to cause him to recommend it most heartily in the extraction of dislocated lenses.

BURNETT.

In SAUNDERS' (151) case the lens was dislocated outwards and forwards, with its outer margin in the anterior chamber pressing on the iris. Acute glaucoma occurred in a few weeks. V, after extraction, rose to $\frac{20}{50}$, with $+ 3$, although perception of light had been abolished. WERNER.

Of THOMAS' (152) 120 extractions, 37 were according to v. Graefe, 82 according to Wecker, and 1 according to Liebreich. Preliminary iridectomy was done in 50 cases. There were 92 successes, 12 partial successes, and 9 failures. In all the later operations antiseptic measures were used. The cases are nicely tabulated. BURNETT.

In WEBSTER'S (153) case the degenerative changes leading to the cataract and its complications were due to sympathetic ophthalmia, and there was only perception of light. On extraction of the calcareous mass, V $= \frac{20}{20}$. BURNETT.

XVI.—VITREOUS.

154. BULL, C. S. Surgical treatment of membranous opacities in the vitreous. *Trans. Amer. Ophth. Soc.*, 1888.

155. HUTCHINSON, J. On certain cases of vitreous opacity in connection with exceptional causes. *The Ophth. Rev.*, 1889, vol. viii., p. 1.

156. HUTCHINSON, J. On a case of hemorrhage into the eye occurring in a young man. *Royal London Ophth. Hosp. Rep.*, vol. xii., part iii., p. 201.

BULL (154) operated 17 times on 15 patients, for breaking up membranous opacities in the vitreous, using sometimes a needle and sometimes a narrow Graefe knife. In 14 cases there was decided improvement in vision, in 3 there was failure. The eye should be free from all irritation before attempting any such surgical procedure. BURNETT.

HUTCHINSON (155) states that the vitreous body usually suffers in conjunction with the retina, while it remains as a rule perfectly transparent, even in long-standing disease of the choroid. In most forms of iritis it scarcely ever suffers, except in the insidious and relapsing form which occurs in young adults, and in iritis due to inherited gout. Simple lowering of the tone of the system commonly gives rise to muscæ, but rarely are the opacities visible with the ophthalmoscope. The author describes one such case, and concludes with the relation of an instance in which the prolonged use of arsenic may have given rise to opacities in the vitreous. WERNER.

HUTCHINSON (156) first refers to the case of repeated hemorrhages in a man named Colin, which he published, in 1881, in the transactions of the Ophthalmological Society. He saw the patient again in 1885 ; the vision was normal, and there had been no recurrence, although constipation still persisted. In 1888 he wrote to say that the attacks had ceased. The author draws attention to the fact that recurrent hemorrhages, usually in both eyes, occur almost exclusively in young men, while single attacks of hemorrhages in middle age commonly affect one eye, and are found in both sexes. Hemorrhagic choroiditis and attachment of the retina are met with under conditions similar to those of recurrent hemorrhage. Seven cases of intraocular hemorrhage are recorded. WERNER.

XVII.—RETINA AND FUNCTIONAL DISTURBANCES.

157. ADAMÜCK. On a case of retinitis albuminurica hæmorrhagica ending in recovery. *Centralbl. f. prakt. Augenhk.*, vol. xiii., p. 98.

158. Alt, A. Detachment of the retina in three successive generations of one family. *Amer. Jour. Ophth.*, Dec., 1888.

159. Berlin, A. On snow-blindness. *Med.-chir. Rundschau*, 1889, No. 2.

160. Berry. Remarks on retrobulbar neuritis, with special reference to the condition of the light-sense in that affection. *Royal London Ophth. Hosp. Rep.*, vol. xii., part iii., p. 244.

161. Capron F. P. Glioma—with remarks. *Trans. Amer. Ophth. Soc.*, 1888.

162. D'Oench, F. E. A peculiar case of detachment of the retina. *N. Y. Med. Rec.*, March 9, 1889.

163. Galezowski. Aperçu général sur le décollement de la rétine et sur sa curabilité. *Rec. a'ophth.*, Feb., 1889, p. 83.

164. Hotz, F. C. Spontaneous cure of a retinal detachment. *Amer. Jour. Ophth.*, Dec., 1888.

165. Kollock, C. W. Amblyopia produced by menstrual suppression. *N. C. Med. Jour.*, 1889.

166. Marlow, J. W. Hysterical blindness in the male. *N. Y. Med. Jour.*, Feb. 9, 1889.

167. Naumow. The question of the cause of congenital amblyopia. *Wjestnik Ophth.*, 1889, No. 2.

168. Sutphen, T. Y. Puncture of the retina for detachment—three operations, with their results. *Trans. Amer. Ophth. Soc.*, 1888.

169. Wheelock, K. K. A case of alcohol and tobacco amblyopia, with remarks. *Jour. Amer. Med. Assoc.*, March 2, 1889.

Adamück (157) reports at length on the rare case of a complete recovery of a man from albuminuric retinitis. Vision had fallen from $\frac{20}{20}$ to $\frac{1}{5}$ in the left and $\frac{1}{10}$ in the right eye, while the general symptoms were well-marked. The ophthalmoscope revealed large hemorrhages, especially at the macula. Baths were prescribed, iodide of potassium given, and in a year all the symptoms had disappeared.

Alt (158) found detachment of the retina in mother, son, and granddaughter. Burnett.

According to Berlin (159), snow-blindness is an affection of the conjunctiva, and not, as was formerly supposed, of the retina. The visual field is not contracted, and there are no scotomas. There is hyperæmia, afterwards chemosis of the conjunctiva, and sometimes corneal opacities. The affection begins with a burning sensation, photophobia, lachrymation, and blepharospasm, which symptoms, however, disappear in about three days after the exciting cause has been done away with. Sometimes secondary hyperæmia of the retina is observed. The cause lies in the mechanical irritation from particles of snow and ice, and in the insolation produced by the sunshine.

BERRY (160) objects in the first place to classifying toxic amblyopia under the heading of retrobulbar neuritis, on the ground that inflammation of the nerve does not account for the constancy in the shape of the scotoma, nor for the fact that the point of greatest saturation in it almost always lies between the point of fixation and the blind spot ; furthermore, the two eyes are equally affected at all stages of toxic amblyopia. In true retrobulbar neuritis, according to the author, the scotoma is irregular in shape and subject to variation, the two eyes are unequally affected, there is no marked preponderance in one sex, the changes in the disc are more pronounced, there is often pain on movement of the eyeball or on pressure. Three cases are recorded showing the diagnostic importance of examining the power of perceiving differences in the intensity of illumination of contiguous objects (L. D.). This was tested with Bjerrum's types (gray letters on gray ground). In the first case, V was normal with Snellen's types, but was reduced to $\frac{4}{70}$ (with L. D. = $\frac{1}{11}$) and an illumination which left the author's V normal. The patient afterwards developed a central scotoma and V became reduced to $\frac{16}{70}$, which disappeared on giving up tobacco. The second patient, a lady, æt. forty, presenting symptoms of retrobulbar neuritis, without scotoma and V = $\frac{20}{20}$, could not see Bjerrum's types (L. D. = $\frac{1}{11}$) even at three feet away. She recovered completely. A third case of obscure nature is also described, in which a defect in the L. D. remained, although marked improvement in vision took place. WERNER.

CAPRON'S (161) case was that of a girl of three years and ten months. At that time the glioma had already invaded the orbit and protruded to the size of a fist. The contents of the orbit were removed. It had been growing less than a year. The child died five weeks after the operation. BURNETT.

D'OENCH (162) reports a case of recent detachment of the retina, in a man of forty-five, involving the upper outer portion of the right eye, in which were found numerous small white dots.

Tn. $V = \frac{1}{\infty}$ in the detached portion. No sugar or albumen in the urine. Burnett.

Galezowski (163) opposes the view that detachment of the retina is the result of a strain upon the retina from the vitreous; he ascribes it to a subretinal serous effusion, the consequence of choroiditis and diminution of tension following liquefaction of the vitreous. Marckwort.

Hotz's (164) patient was a man seventy-four years old, on whose right eye he had operated for cataract. With the left he could never see well, being, as he thought, very nearsighted in it. Hotz found a detachment of the retina, — T, lens and vitreous clear, $V = \frac{1}{\infty}$ in lower part of field. On removing the bandages after the cataract operation it was found that the detachment in the left eye had disappeared, and with — 4 $V = \frac{20}{70}$. A year later the same satisfactory condition was found. Burnett.

Kollock's (165) case was that of a negro girl of thirteen who had just begun to menstruate. Menstruation ceased, and for three months there was total blindness in the right and nearly so in the left eye. No organic change in the media or at the fundus. V was restored on resumption of the menstruation. Burnett.

Marlow's (166) case was that of a man of twenty-eight, below medium height and physique. No recent illness or nervous complaint. Sudden and complete blindness after feeling as if something had gotten into the eye. He recovered the next day on having atropine instilled into the eyes. Burnett.

Under this title, which does not exactly correspond to the contents, Naumow (167) presents a review of the anatomical examination of the eyes of forty-seven children who had died during or soon after birth. In one case he found œdema of the retina; in another, choked disc; in six, hemorrhages at the macula lutea; in one, hemorrhage into the retina and choroid; in one, hemorrhage into the choroid corresponding to the region of the yellow spot (in one eye); in two, œdema of the macular lutea. In the eyes of children born too soon (twenty-two pairs of such eyes) he did not find any pathological changes. The changes described were found mostly in first-born children, or children of great weight, after prolonged labor, in narrow pelvis of the mother. The cause must be sought in the increased intracranial pressure, due to pressure upon the head, or in congestion of the head resulting from pressure upon the neck (exerted by the genital passage or the encircling umbilical cord). Hirschmann.

Sutphen (168) resorted to puncture of the retina through the sclera. Three operations were done in all, and both eyes were saved, with good vision. The myopia was of low degree.

Burnett.

The peculiarity of Wheelock's (169) case consisted in the fact that there was no loss of color-perception nor well-marked central scotoma. V was reduced to $\frac{1}{10}$, but was restored to the normal on abandonment of the tobacco and alcohol (beer) and injection of strychnine. Burnett.

XVIII.—OPTIC NERVE.

170. Derby, R. H. Case of monocular neuroretinitis. *Trans. Amer. Ophth. Soc.*, 1888.

171. Randall, B. H. Anomalies of the veins upon the optic disc. *Trans. Amer. Ophth. Soc.*, 1888.

172. Randall, B. A. Cases of outgrowth on the optic disc. *Trans. Amer. Ophth. Soc.*, 1888.

173. Richter. Pathologico-anatomical and clinical notes on the conduction of light in the human brain. *Arch. f. Psych. u. Nervenkrankh.*, vol. xx., No. 2.

Derby's (170) case of monocular neuroretinitis was that of a girl of twelve who had never menstruated. She had no history of syphilis, and was otherwise healthy. Burnett.

The anomalies figured by Randall (171) are: (1) Bifurcation of the retinal veins in their passage towards the disc; (2) vascular circle formed by a retinal vein; (3) an anastomotic branch uniting the central with an aberrant vein. Burnett.

Randall (172) gives drawings of two small cysts growing out from a vessel on the optic disc, which he thinks may be the remains of a hyaloid artery, and four others of large, dense white masses. Burnett.

Richter (173) reports on two autopsies, which add further proof of the assertion that occasionally monocular or binocular atrophy of the optic nerve due to a cerebral affection, though it may have existed but a short time, can be traced macroscopically beyond the tractus. In other cases no signs of it can be found even after ten years and more. In a paralytic who had been blind for twenty-four years he only found a flattening of the corpus geniculatum externum, while in a taboparalytic woman who had been blind six years there was atrophy of the tractus, the corpus geniculatum externum, and the pulvinar.

XIX.—INJURIES, FOREIGN BODIES (PARASITES).

174. JACKSON, E. Bruise of the eyeball, localized œdema of the choroid and retina, transient astigmatism. *Trans. Amer. Ophth. Soc.*, 1888.

175. MCKEOWN. Foreign bodies in the eye. *Brit. Med. Jour.*, 1889, p. 387. (Extraction of a chip of steel with good result.)

176. PHILLIPS, W. A. The electro-magnet. *Jour. Ophth. Otol. and Laryng.*, Jan., 1889.

177. REECE, R. A piece of glass in the eyeball for seven years and ninety-four days. *St. Barthol. Hosp. Rep.*, vol. xxiv., p. 201.

178. TROMPETTER. Case of removal of a chip of steel from the eye with the electro-magnet. *Zeh. klin. Monatsbl.*, vol. xxvii., p. 87. (Successful extraction with $V = \frac{15}{30}$.)

JACKSON'S (174) paper is accompanied with two very excellent chromo-lithographs representing two stages of the œdema of the retina and choroid following a bruise of the eyeball. The transient astigmatism amounted to about —0.75, ax. 180°.

BURNETT.

PHILLIPS (176) relates three cases in which he used the magnet for removal of foreign bodies from the interior of the eye. (1) Bit of steel in the fundus, which could be seen on the retina downward and outward from the disc. Gruening's (permanent) magnet was introduced through an opening made in the sclera, and the bit of steel removed. But little reaction followed, and the immediate result was good. The eye finally succumbed to an attack of chorio-cyclitis after a severe cold and intermittent fever. (2) A bit of steel which had passed through the cornea and lens was searched for with the electro-magnetic needle, and successfully extracted. On the morning of the third day after the operation the iris was a little inflamed. The greater part of the lens was removed, followed by a rapid recovery, and $V \frac{20}{40}$. (How long after the operation ?) (3) A bit of steel lodged in the iris and lens ; removed with the aid of the electro-magnet.

BURNETT.

REECE (177) records the case of a myopic student whose eye was injured in 1880 by a stone breaking his glasses. He was subsequently operated on for traumatic cataract in 1881 and 1882 by Teale. The eye then remained in a quiescent condition until 1887, when it was excised on account of photophobia and lachry-

mation. On opening it a piece of glass was found behind the iris in contact with the ciliary processes at the lower part of the eye.
WERNER.

XX.—OCULAR AFFECTIONS IN CONSTITUTIONAL DISEASES.

179. ADAMKIEWICZ. Unilateral progressive paralysis of the cerebral nerves. *Wiener med. Wochenschr.*, 1889, No. 1.

180. ANDERSON. Some ocular and nervous affections in diabetes and allied affections. *Ophth. Rev.*, vol. viii., pp. 37 and 65.

181. FREUND. On optical aphasia and mental blindness. *Arch. f. Psych. u. Nervenkrankh.*, vol. xx., Nos. 1 and 2.

182. GOULD, GEO. M. Three remarkable cases of reflex neurosis due to eye-strain. *Med. and Surg. Rep.*, Feb. 9, 1889.

183. HUTCHINSON, J. Amaurosis (without optic neuritis) due to cerebral tumor. *Royal London Ophth. Hosp. Rep.*, vol. xii., pt. iii., p. 258.

184. KUNN. Case of mydriasis spastica and neuroretinitis, ending in atrophy of the optic nerve in tetanus. *Wiener klin. Wochenschr.*, No. 12.

185. MONEY, A. On the dilatation of the pupil in locomotor ataxy. *The Lancet*, Jan., 1889, p. 170.

186. NOTHNAGEL. On tumors of the corpora quadrigemina. *Wiener med. Wochenschr.*, 1889, No. 3.

187. SATTLER. On herpes zoster ophthalmicus. *Wiener klin. Wochenschr.*, 1889, No. 7.

188. SEGGEL. Ophthalmoscopic signs of brain-syphilis. *Deutsches Arch. f. klin. Med.*, No. 4, p. 423.

189. TEPLJASCHIN, A. Cataract resulting from chronic poisoning with secale cornutum. *Congress of Russian Physicians*, 1888.

190. UHTHOFF. On incipient multiple sclerosis. *Berliner Gesellsch. f. Psych. u. Nervenkrankh.*, *Report*, 1889.

191. WERNICKE. Circumscribed affection of the lower parietal lobules. *Arch. f. Psych. u. Nervenkrankh.*, vol. xx., No. 1.

192. ZIEM. Limitation of the visual field in affections of the nose. *Wiener klin. Wochenschr.*, 1889, No. 12, and *Deutsche med. Wochenschr.*, No. 5.

ADAMKIEWICZ (179) reports the case of a woman aged 54, who for five months had suffered with right-sided headache, in whom the abducent and trochlearis nerves of the right side were completely and the oculomotor partially paralyzed. Sensibility and taste on that side were absent, sight, hearing, the muscles and mental powers intact. No reflex lachrymation. Adamkiewicz assumed a thickening of the periosteum at the point of exit of the nerves at the base of the brain. The autopsy revealed a carcinoma at that spot. In spite of the absence of tears, the eye showed no symptoms even until death of a neuro-paralytic nature.

ANDERSON (180) contributes two papers containing general remarks on diabetes and cases illustrating the occurrence of retinitis, ocular paralysis, local neuralgia, and multiple peripheral neuritis. In the two cases of retinitis, the vitreous humor was free from hemorrhages : in the first case the retinal hemorrhages and white patches were not unlike those of albuminuric retinitis, except that the blood extravasations were mostly circular and lying under the vessels, and the patches were not so clear white and were more irregularly scattered. WERNER.

Under the name of optic aphasia FREUND (181) describes a disease characterized by very marked difficulty in finding nouns for concrete ideas, and by the simultaneous existence of cerebral optical disturbances. The patient cannot find the proper word when objects are shown him, although he knows them, but readily names them when he closes his eyes and touches them, when sensations of touch are thus excited. This state must not be confounded with mental blindness, in which the patient sees every thing, but in which the impressions on his memory have faded. The former is due to lesions in the cerebral optical conducting apparatus, which can anatomically be demonstrated. It may manifest itself in three combinations : (1) optical aphasia with hemianopsia ; (2) with mental blindness ; (3) with mental blindness and severe sensory acoustic aphasia. For details see the clinical histories and reports of the autopsies.

The reflex neuroses due to eye-strain reported by GOULD (182) are as follows : (1) Chorea of several years' duration ; relieved at once by the use of a glass of + 1. ⊃ + 0.75, 90°. (2) Flatulent dyspepsia of twenty years' standing ; cured by wearing — 0.25 ⊃ + 0.75, ax. 110° R, — 0.25 ⊃ + 1., 70° L. (3) Cardiac palpitation, etc., relieved by correction of astigmatism. BURNETT.

HUTCHINSON (183) records the case of a man aged twenty, who suffered from marked amblyopia, with dilated, sluggish pupils, associated with headache and occasional vomiting. There was a definite central scotoma for red and green. After death, which occurred suddenly, a soft glioma was found at the apex of the right temporo-sphenoidal lobe. The discs and optic nerves were normal, but the perineural sheath was filled with small cells and delicate connective tissue, apparently gliomatous. The author thinks it possible that optic neuritis might have developed had the patient lived longer. WERNER.

KUNN (184) cites the mydriasis and neuroretinitis as proof that tetanus is a central affection located in the cervical part of the spinal cord. According to him mydriasis is observed in every case of inflammation of that part of the cord, retinitis, however, but seldom, though it had been noticed in chronic and acute myelitis, in transverse myelitis, and in caries of the cervical vertebræ.

MONEY (185) has noticed dilatation of the pupils in cases of locomotor ataxy when the light was intense, *e. g.*, in the ophthalmoscope room. He thinks that the heat and light probably act on the conjunctiva and produce reflex sensory dilatation. The pupils contracted on convergence, but did not react to light and were not very small. (Nothing is stated regarding the vision of these patients, and the artificial light is not more intense than diffused light on a bright day.—L. W.) WERNER.

In a boy aged fifteen, who had fallen from a tree and apparently had escaped unhurt, NOTHNAGEL (186) observed after a while unsteady walk, followed by symptoms of intracranial increase of pressure, particularly choked disc. There were, however, no symptoms of circumscribed cerebral affection. Besides the ataxy there was rigidity of the eyeballs to the left and upwards, and total paralysis of the abducent and partial of the oculomotor nerves. The pupils, of equal diameter, responded slowly. The diagnosis of a tumor of the corpora quadrigemina with secondary hydrocephalus was confirmed by the autopsy.

In SATTLER'S (187) case the herpes in the first branch of the fifth nerve had developed after poisoning with carbon dioxide gas The man, who was eighty-five years old, died two weeks later. An inflammatory infiltration of the interstitial tissue and disappearance of the ganglion cells were found in the medial part of Gasser's ganglion, which corresponds to the first branch. The fibres connected with the ganglion cells had degenerated, which degeneration could be traced through the ciliary ganglion into the globe. The nerve-fibres passing through were not affected. A mycotic origin of the disease (Erb) could not be demonstrated.

From his observations SEGGEL (188) concludes that only those forms of retinitis are characteristic of a simultaneous disease of the cerebral blood-vessels in which the affection of the vessels takes the form of bright deposits in their walls or conversion into bright stripes with more or less marked obstruction of the calibre. These changes in the blood-vessels, found in patients who had no visual disturbances, but presented symptoms of cerebral syphilis, disappeared under antisyphilitic treatment. The cases belonged to a later stage of syphilis. To assume, however, an affection of the blood-vessels merely from the appearance of small clustering deposits near them (Ostwald) he does not consider justifiable as yet.

In the winter of 1879–80 TEPLJASCHIN (189) observed an epidemic of raphania, of a convulsive type exclusively, mostly in younger people. Beginning with 1882, cataract patients between the ages of thirty and forty began to appear, who had suffered with convulsions. The cataract appeared on both eyes, was moderately soft, and had a larger or a smaller nucleus, according to the age. It had required from two to seven years to ripen. The cause of this development of cataract after raphania Tepljaschin seeks in the disturbance of the nutrition of the lens, produced either by the convulsions (the significance of which in the etiology of cataract of adults Logetschnikow first pointed out), or by the spasm of the blood-vessels. HIRSCHMANN.

UHTHOFF'S (190) patient showed discoloration of the temporal half of the papilla, especially in the right eye, with sudden very marked diminution of sight, while the left was only gradually affected. At the time of presentation of the case there was a small ring-scotoma for red and green in both eyes.

WERNICKE (191) observed a young man aged twenty, who, aside from some symptoms resulting from several apoplectic attacks which manifested themselves later, held his head and eyes turned to the right (conjugate deviation of the head and eyes) after a slight apoplectic attack. There was also slight paralysis of the facial nerve, diminution of hearing on the left side, reduction of vision without a defect of the visual field, anæsthesia of the left cornea, slight paresis of the left extremities, with complete hemianæsthesia. Death three days later in consequence of further apoplexies. The deviation of the eyes could be attributed to a fresh lesion occupying precisely the cortex of the lower parietal lobule (angular gyrus) of the right side. The hemianæsthesia was ascribable to the simultaneous affection of Charcot's carrefour sensitif, while the hemiparesis was an indirect result of the lesion. Destruction of both parietal lobules must therefore simulate ophthalmoplegia externa.

Ziem (192) reports four cases in which contraction of the visual field, pains in the forehead and temple, clouded vision, and the appearance of spots in the visual field were improved or removed by treatment of the nose with the galvano-cautery.

MISCELLANEOUS NOTES.

The Königsberg ophthalmologist, Julius Jacobson, died on Sept. 15, 1889, after a prolonged illness. He was born at Königsberg, Aug. 18, 1828, studied there, at Berlin, and at Prague, passed his examination at Königsberg in 1853, and began the practice of medicine in that city. In 1857 he became "Privat docent" for ophthalmology, was appointed Professor extraordinarius in 1859, and Professor ordinarius in 1872. From the time he was a student he attached himself to the circle of A. v. Graefe, and remained a true friend to him beyond the latter's grave. It is his particular merit that he succeeded in separating ophthalmology from surgery, and the erection of special ophthalmic clinics at the Prussian universities, for which he earnestly labored, is due to him. A number of the most prominent oculists, among others A. v. Hippel, Samelsohn, Vossius, Treitel, Alexander, are proud of having been his scholars. Of the numerous publications of Jacobson the following in particular may be cited: "On Syphilitic Retinitis; Diphtheria of the Conjunctiva" (*Königsberger med. Jahrb.*); A New and Safe Method for the Extraction of Senile Cataract" (1863); "Annual Report of the Königsberg Ophthalmic Clinic from 1877 to 1879" (Berlin, 1880); "Cataract Extraction with Flap Section" (*v. Graefe's Arch.*, vol. xi.); "On Graefe's Latest Method of Extracting Cataract" (*ibid.*, vol. xiv.); "Clinical Contributions to the Knowledge of Glaucoma" (*ibid.*, vol. xix.); "Preparatory Iridectomy and Antisepsis" (*ibid.*, vol. xxx.); "Albrecht v. Graefe's Importance for our Science as Shown from his Works" (Berlin, 1885).

Prof. Dr. ZEHENDER, of Rostock, retired at the end of the summer term, having completed his seventieth year. Prof. RUDOLF BERLIN, of Stuttgart, has taken his place at the University. Prof. SCHLEICH, of Tübingen, has taken charge of the latter's ophthalmic institute, as well as of the professorship for ophthalmology, at the Stuttgart Veterinary School.

Privat docent Dr. MAX KNIES, of Freiburg, i. B. has been appointed Professor extraordinarius.

The prize offered by Prof. v. WELZ for the best paper published in *Graefe's Archives* between 1884 and 1886 has been awarded to Professor Dr. Deutschmann, of Hamburg, for his paper entitled, "The Pathogeny of Sympathetic Ophthalmia (Ophthalmia Migratoria)." The paper of Dr. Uhthoff (Berlin): "Investigations on the Influence of Chronic Alcoholism on the Human Visual Organ" received honorable mention.

No meeting of the Ophthalmological Society will be held at Heidelberg in 1890. A numerous attendance of the members, however, is requested at the International Medical Congress, which will meet at Berlin from Aug. 4th to Aug. 9th.

Dr. EDWARD JACKSON, of Philadelphia, enters into the editorial staff of the *Ophthalmic Review*, in place of Dr. James Anderson, of London.

Prof. A. v. HIPPEL, of Giessen, has been appointed to succeed the late Prof. JUL. JACOBSON in the chair of ophthalmology at the University of Königsberg, Prussia.

Dr. DAVID CLARK COCKS, Surgeon to the New York Eye and Ear Infirmary, died from the consequences of influenza, January 14, 1890, in the thirty-ninth year of his age. He was a competent and industrious ophthalmic and aural practitioner, and has published several valuable contributions to practical eye surgery. His memory will be kept in honor by his New York colleagues.

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ARCHIVES OF OPHTHALMOLOGY.

TUBERCULOSIS OF THE CONJUNCTIVA.

By Dr. SWAN M. BURNETT, Washington.

(*With a heliotype plate, containing five figures.*)

AN examination of the literature of the subject shows that tuberculosis may affect every tissue of the eye—the iris, choroid, ciliary body very commonly; less frequently, the cornea (Sattler, 44[1]), retina (Manfredi, 30), and optic nerve (Weiss, 52; Sattler, 44), and (secondarily) the sclera, and the conjunctiva. The frequency of its occurrence in the eye as compared with other affections has been estimated very variously. Some—as Hirschberg—place it as high as 1 : 6000, while others—as Mules—put it as low as 1 : 30,000; but even the highest estimate would make it far from a common occurrence. Of the eye-tissues, statistics show that the most frequent seat of tubercle is the uveal tract—iris and choroid,—while its least frequent occurrence is in the optic nerve and retina. Even up to 1867 the conjunctiva was thought, on the authority of Virchow (49), to have an immunity from tubercular disease. This was at a time, however, when there was thought to be a difference, pathologically, between lupus and tuberculosis, for lupus of the conjunctiva is mentioned by some of the writers before that time. The identity of lupus and tubercle is still denied by many, notwithstanding the fact that the essential pathological characters—giant-cells and bacilli—are the same in both. Neumann (33) makes this distinction, that the tubercle breaks down into a cheesy mass, while lupus does not; and cases of lupus of the conjunctiva have always been found, he says, associated with lupus either in the skin of the lid or other part of the body. It would seem, however, from the experiments of Trousseau (47), and Pagenstecher and Pfeiffer (34), that inoculations of the anterior chamber of rabbits with lupus from the human conjunctiva give rise to the same kind of pathological change in the iris as inoculation with genuine tubercle. This important fact, however, is to be noted, that in none of these cases has a general tuberculosis been produced. The general drift of opinion seems to be toward a pathological identity of the two affections, with perhaps some differences in the clinical manifestations. As a contribution to the further study of the subject I offer the following:

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[1] These numbers refer to the bibliography.

Leroy Johnson, a colored boy of fifteen years, was first seen by me at my clinic on the 29th of July, 1887. He was of a healthy but not robust appearance. No very satisfactory antecedent history could be obtained, but it was evident that he had been troubled with his eyes for at least three or four years. He had never had any serious illness, but the observation of a large scar on the left cheek, three by four inches, the lower border following the ramus of the inferior maxilla, led to the admission that he "had had scrofula" some years ago. The scar had not led to any contraction of the tissues in the vicinity, but is smooth with irregular edges, and on its lower margin there are four hard, smooth tumors which resemble the keloids to which this race is very subject. At that time the following notes were taken: L. symblepharon of both lids, the cornea not visible, being covered by the soft thickened upper lid. The lower lid was also thickened and the conjunctiva, of which a portion could be brought to view, was irregular and granular on its surface with here and there defects in its substance. What corresponded to the retrotarsal fold came up to the very margin of the cornea and was thickened and nodular. The condition of the conjunctiva of the R was the same as that of the L, only much less pronounced. The cornea could still be seen by pulling back the thickened upper lid. The corneal surface was hazy, with some vessels running over it, and vision was very much reduced. There was no considerable discharge of any kind, but strings of thick mucopus lay in the crevices of the granulations and on the edges of the lids. There was no tendency to entropion; an enlarged gland was felt in front of the ear, on the right side, but not on the left. There was no pain complained of, nor had there been at any time. A bit of conjunctiva was cut from the lower lid of the left eye and put aside for examination. On inquiry it was learned that he was one of a family of six children, all of whom were said to be healthy. The father and mother were living, and none of their children had died.

The boy did not return until eighteen months later, when the photograph, of which Fig. 1 of accompanying plate is a copy, was taken, and the drawings, Figs. 4 and 5 were made. It will be seen from an examination of these that there is complete symblepharon on the left side, the margins of the lids coming in some places quite together. The integument, and even the edges of the lids, are unaffected, and the normal directions of the cilia are not materially altered. No portion of the eyeball was visible, but it could be felt through the soft, thickened lids, as a doughy, ill-defined mass. The symblepharon in the right eye was not so extensive, but both retrotarsal folds were entirely abolished. The thickened and nodular conjunctiva came up to and encroached upon the cornea both above and below. The corneal surface, which was still visible after the thickened upper lid was pressed back, was grayish, with thickened epithelium, and the color of the iris was barely visible through it. He could, however, count fingers at one metre when the upper lid was raised. His general health seems to be perfect. Dr. Murray, director of the clinic for diseases of the throat and chest, was kind enough to make a careful examination of his chest, and reports that he finds nothing beyond a few moist râles at the posterior portion on the right side. There is no consolidation anywhere.

No treatment has been instituted beyond a wash of mercuric bichloride, 1 : 10,000. Dr. Wm. M. Gray of the Army Medical Museum, made sections of the bit of excised conjunctiva, a photograph of one of which is shown in Fig. 2. The giant-cells are shown here with great clearness. A piece, excised later, was examined for the tubercle bacillus, and some were found in the tissue. A scraping of the surface of the diseased tissue contained many bacilli, and a photograph of a specimen, also made by Dr. Gray, is shown in Fig. 3. From a histological standpoint the genuine tuberculous character of the affection is clearly established, and the clinical history warrants the opinion that it is localized and primary.

A reference to the bibliography appended to this paper will show that the literature of tuberculosis of the conjunctiva is by no means scanty; and yet even the most recent text-books mention it either casually or not at all. The lamented Horner, in whose clinic the affection has been studied more closely than elsewhere, has considered it in his part of the treatise on diseases of the eye in childhood, in Gerhard's "Handbuch"; and Amiet's thesis (3) contains the fullest account of cases on record, up to the date of its publication. Neither Wecker (1889) nor Galezowski (1888) give cases. It is not mentioned by Sämisch, in his treatise on diseases of the conjunctiva, in the "Gräfe-Sämisch Handbuch," except as lupus. Schmidt-Rimpler calls attention to it, but says it never occurs spontaneously. Fuchs gives it considerable attention. Mooren, in his statistics of 100,000 eye cases, does not record a single instance.[1] The recorded cases are so widely scattered throughout the periodical literature of general and special medicine that we have thought it not without value to the future student to give a *résumé* of the more important facts in such articles as we have been able to consult.

ALEXANDER (2). Man, twenty-seven years, tumor of lower lid like an enlarged tarsus, conj. thickened and granular, retrotarsal fold ulcerated, a probe passing through to the bone. Conj. bulbi below in same condition up to corneal edge; remaining parts of eye intact; vision not affected.

AMIET (3). Gives the history of two cases from Haab's clinic. Case 1.—Woman, twenty-seven years; left eye. Nodules on conjunctiva of both lids, and at caruncle. Right eye normal; not seen for two years; the upper lid hung down, but not red or swollen. Pre-auricular and submaxillary glands swollen. Ulcer on conjunctiva, on temporal side of globe; retrotarsal folds thickened, granular and nodular, almost fungus-like. Upper portion of cornea pannous; anterior chamber and iris normal. Diseased tissue extirpated, and wound cauterized with platinum wire. Healing good. Some bacilli found in the excised tissue. Case 2.—Girl, of twenty-one. Outer part of lower lid everted and nodular ; upper lid normal. Lower retrotarsal fold affected ; pre-auricular and submaxillary glands swollen. Child healthy. Removal of diseased tissue, with knife and cauterization ; return, and again removed. Healing good. Excised pieces showed tubercular nodules and some bacilli. His experiments in the inoculation of the conjunctiva of rabbits were negative. From a study of forty-seven cases recorded up to that time, he concludes : (1) That tuberculosis of the conjunctiva occurs from the tenth to the thirtieth year ; (2) generally affects women ; (3) both eyes affected about equally ; (4) seldom binocular ; (5) conjunctiva palpebrarum its most frequent seat ; (6) upper lid most frequently affected ; (7) cornea often pannous ; (8) swelling of pre-auricular and submaxillary glands nearly always present ; (9) healing can be effected through early and prompt treatment, but there are often recurrences, and when the disease is extensive not much should be expected ; (10) treatment must be surgical, removed by sharp spoon, knife, or cautery.

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[1] Noyes, whose text-book (Wm. Wood & Co., N. Y., 1890) has appeared since this paper was placed in the hands of the printer, gives quite a long paragraph to it.

ARLT (4). First noticed and diagnosticated primary lupus of the conjunctiva, originating in the retrotarsal fold. It afterwards invaded the bulbar conjunctiva and cornea and lid, and later the skin of the cheek and alæ nasi.

BAUMGARTEN (5). Case 1.—Girl, of sixteen years. Ulcers of conj. of globe of both eyes encroaching on the cornea in L. Palp. conj. nodular. Diseased tissue removed with a sharp spoon ; partial ankylo-symblepharon in L. Atrophy of globe, R. Enucleation of L. Section showed conj., iris, and cornea affected with tuberculosis. Case 2.—Girl, of nine years. Conj. of lids of both eyes nodular.

CSAPODI (8). Case 1.—Girl, of sixteen. Scrofulous ; right eye : small ulcer on conjunctiva of lower lid ; no bacilli found. Case 2.—Girl, ten years. Scars on neck, and lupus of nose. Both eyes affected ; partial symblepharon in both lower lids ; palpebral conjunctiva, thickened and nodular ; conjunctiva bulbi also thickened, and the cornea invaded. No bacilli found.

ELSCHNIG (10). Man, of thirty years. Began a year before. Right upper lid thickened ; conjunctiva swollen and covered with numerous granulations and excrescences, with a tendency to ulceration. Bacilli found in tissue and scrapings of the surface.

FALCHI (11). Boy, of seven years, not robust. Ragged ulcer on conjunctiva of left lower lid ; also nodules, not painful to touch; slight ectropion. Affected part removed; good cicatrization. Giant-cells found in the removed tissue.

GAYET (14). Woman, twenty-six; right eye: conjunctiva of lid; pre-auricular glands enlarged on that side; nodules removed, and inoculations made in guinea-pigs and rabbits with success. The condition of the eye became aggravated, lid adhered to the ball, the cornea became involved, and the eye was removed; giant-cells and bacilli found in the diseased tissue. Is of the opinion that it was an initial lesion.

HILL-GRIFFITH (16). Boy of four years; right eye: entire ocular conjunctiva thickened and studded with yellowish-white infiltrations; felt doughy to the touch; no pain; conjunctiva of lids and retrotarsal folds unaffected; other parts of the eye healthy; pre-parotid and submaxillary glands swollen on that side. Abscesses in neck and at outer commissure of the eye formed an ulcer which was thought to be lupus. No bacilli were found, but giant-cells were.

Little had seen a case somewhat similar to the above in the conjunct. bulbi left eye of a man of thirty-five years.

HAAB (17). Some of these six cases are probably among those that have been published elsewhere as coming from the clinic at Zürich. Case 1.—Man of thirty-two years; both eyes confined to conj. of lid. 2.—Woman, eighteen years; conj. lid of R eye. 3.—Woman, thirty-three; L eye, conj. of upper lid. 4.—Girl of twenty-two; conj. of L upper lid. 5.—Girl of fourteen; conj. and retrotarsal folds upper and lower lids, L. 6.—Girl of seventeen years; conj. of both lids, L. Giant-cells found in all. In all cases there was enlargement of pre-auricular glands. He thinks with Arlt that lupus has a tendency to pass over to the integument.

GERIN-ROZE (15). Man of thirty-four years; conjunctiva of both lids affected; phthisis of lungs and larynx.

FANO (12). Girl of thirteen; operated on for squint on 31st August, 1880; suture used; good healing, with no remaining hyperæmia of conjunctiva. Two months after, a gray nodule at superior internal part of the globe, which increased in size. A month afterwards the eye was enucleated, and giant-cells found in the conjunctival nodule.

HERTER (19). Grayish-yellow nodules, lower retrotarsal fold, and conjunctiva on one side. Giant-cells found.

RAMPOLDI (41). Man of eighteen years; tuberculosis of apices of lungs; ovoid ulcer on conj. of left upper lid; no giant-cells or bacilli found; removed by knife and cautery. Six months later it returned, and the patient died. No bacilli found in the conj., but plenty in the lungs.

HOCK (20). Case 1.—Boy of two years; ulcer of conj. right eye near outer angle, which began as a small nodule; went on to perforation of globe, and the child died. 2.—Man of sixty-two; ulcer of conj. upper lid outer angle of right eye. Died a few weeks after he was first seen from phthisis.

HIRSCHBERG (21). Girl of four years; ulceration of bulbar conj., retrotarsal fold, and part of the palp. conj.; cornea punctate; submaxillary gland swollen; removal; no return after three and a half years.

v. MILLIGAN (29). Girl of eleven years; conj. lower lid R eye; began as a chalazion. A year after there were nodules on the conj. of ball and lower lid, the ulceration passing over to the integument of the lid in one place; swelling in front of R ear; scraped off diseased tissue; giant-cells found. The only case in twenty thousand eye cases.

KNAPP (22). Boy of nineteen; no history of tuberculosis in family; lupus of alæ nasi; R eye healthy; L cornea and bulbar conj. healthy; nodules on palp. conj. of upper and lower lids, and some small ulcers; two large ulcers near edge of upper lid; tubercle bacilli found in excised tissue, and inoculations in ant. chamb. of rabbits were successful.

KOSTER (23). This is the first reported case of tuberculosis of the conj. It was a tumor, ulcerated, which affected the conj. of lower lid, and passed back, involving the retrotarsal fold.

LUC (24). Case 1.—Girl of eighteen; scrofulous; lupus of right nose; tumor on conjunctiva of R lower lid. It was scraped, and there was no return after two years. 2.—Lupus of nose, lips, and conjunctiva bulbi right in a girl of seventeen. 3.—Lupus of face propagated to conjunctiva in a man of twenty-five. 4.—Large lupus of nose implicating lids and conj. R in a girl. He thinks conjunctival tuberculosis is associated with tuberculosis elsewhere; lupus, with struma; pre-auricular enlargements in tubercle, none in lupus; course rapid in tuberculosis, slow in lupus.

Leidholdt (26). A man of fifty years, no evidence of tuberculosis elsewhere, had ulcers on the palpebral conj. and retrotarsal fold which contained the bacilli. Eye and eyelids finally removed.

Löwenthall (27). A woman, who had nursed a phthisical husband; developed, nine weeks after his death, like trachoma, which resulted in destruction of conjunctiva and cornea.

Manz (32). Boy, two and a half years; conj. of both lids of R eye thickened and nodular; edge of lower lid destroyed at its outer half; two ulcers on upper lid; conj. bulbi also affected; pre-auricular and submaxillary glands enlarged. Child died of tubercular meningitis.

Pagenstecher and **Pfeiffer** (34). Made inoculations of anterior chamber of rabbits from a case of conjunctival tuberculosis successfully; also made successful inoculation from lupus of nose and conjunctiva. The conclusion is that lupus is a localized tuberculosis of the skin.

Parinaud, H. (36). Girl, seventeen and a half years; ulcer conjunctiva left upper lid six *mm* long, irregular, granular, and with whitish-yellow nodules; enlargement of pre-auricular gland; had existed for three months. No history of tubercle in family, and no evidence of any in herself. Inoculation of anterior chamber of rabbits produced tuberculosis of iris and lungs.

Peck, E. S. (37). Girl nine years; ulcer, left upper lid, seven *mm* long by three *mm* broad, ovoid in shape, extending from inner canthus; granular floor, and bled easily; whitish secretion; ocular conjunctiva not affected; patient otherwise healthy; cauterized with argent. nit.; healed. Two months afterward another ulcer developed in the middle portion of the same lid, which was also healed by cauterization. Five months afterwards another ulcer at inner canthus, which invaded the integument, and a fourth at the time of report.

Pflueger (39). Girl of twenty years; left upper lid thickened and hanging down; on conj. an ulcer 11 x 2 *mm;* globe free; ulcer behind the left ear appeared after the conjunctival ulcer; cauterized; healed. No microscopical examination made.

Rhein (40). Case 1.—Man of twenty-two years; L eye, total symblepharon; R conj. nodular and ulcerated in places; cornea perforated; bacilli found in discharges; successful inoculations. 2.—Girl of nineteen; L eye, both upper and lower lids nodular, with swollen conjunctiva; bulbar conjunctiva slightly so; pre-auricular glands swollen ; bacilli found in extirpated tissue, and inoculations were successful.

Sattler (44). In the case published in *Irish Med. Gazette* (45) he made sections of the lids, and found tubercle in the conj., subconj. tissue, and tarsus. In a more recent case a woman of fifty-six years had a tumor on the cornea, like proud-flesh, which spread to the conj. On examination after removal the characteristics of tubercle were found in the conj. and cornea.

Stœlting (46). Case 1.—Girl of nine years ; ulcer on conjunctiva R upper lid ; granular growths at bottom, and slight corneal pannus ; pre-auricular glands on that side swollen ; mass removed, and bacilli found ; recovery apparently complete. 2.—Girl of twenty years ; typical tubercular ulcer of left cheek ; lungs normal ; double dacryocystitis, which was treated. Some months later conjunctiva of L upper lid developed an ulcer near the retrotarsal fold ; granular at bottom ; conjunctiva nodular ; pre-auricular glands enlarged. Professor Horner removed the diseased mass, and rabbits were inoculated in the anterior chamber successfully. R eye became affected some months later. There was a recurrence in the left eye. 3.—Girl of sixteen ; ulcer on conj. of left lower lid, with granular bottom ; pre-auricular glands enlarged on that side ; no tuberculosis of lungs detected ; bacilli found in tissue removed by Professor Horner, and inoculations were successful.

Walb (51). Boy. Upper and outer part of the bulbar conj. of R eye had an ulcer which finally led to perforation of the sclera.

Walb (51*a*). A case additional to (51), child one and one half years. L eye : lids thickened, but no irritation of the eye. Thickened secretion on edges of lids. Conj. on both lids thickened with defects and nodules. Pre-auricular glands swollen and fluctuating. Cauterized the diseased parts with success. He considers that eye tuberculosis may be primary.

The important practical question in connection with tuberculosis of the conjunctiva as well as with other localized tuberculosis of the eye is, as to whether it ever is, or can be, primary, and whether the removal of the diseased part will prevent a general infection. An examination of the clinical histories of the recorded cases seems to show that while in some instances the conjunctival affection was evidently secondary and in many others doubtful, there are still some cases where we must accept the conjunctival affection as the primary lesion. The proof of this is the lack of evidence of tuberculosis elsewhere and the non-appearance of the disease in other parts of the body after removal of the diseased tissue. And, after all, the real wonder is, that we do not have more frequent inoculations of the conjunctiva than we do. The experiments of Valude (48*a*) show that while the intact healthy conjunctiva may not be inoculated with tuberculous bacilli, it is, on the other hand readily inoculated when abraded. Fuchs (13) also holds the same view. Now of all the mucous membranes, there are probably none more liable to abrasions and epithelial defects than the conjunctiva, particularly in childhood and among those who are from faulty nutrition and general tendency to tuberculous disease, most liable to infection of this character. Phlyctenular conjunctivitis is the most frequent eye-disease among strumous children. When these vesicles rupture and form ulcers, they make a most favorable nidus for the bacilli, which may be conveyed into the eye with the dust in the air, or by other means, as the towels and handkerchiefs used by phthisics. And it would seem, all things considered, that this explanation of direct inoculation[1] is much more reasonable than the supposition of a secondary infection through the circulation—though tuberculosis of the interior of the eye cannot be thus accounted for so satisfactorily, except in cases of injury. I have seen several times, in young people, ulcers of the conjunctiva near the lid margin, which had all the macroscopical appearances of tubercular ulcers, and which disappeared after thorough cauterization with the pure nitrate-of-silver stick. That such ulcers may even heal spontaneously, under good hygienic management does not seem to me improbable.

When the tubercle is situated where the complete removal is possible without material injury to the visual organ, there can, of course, be no question as to the desirability—the necessity indeed—of total extirpation ; and where the function of the organ is already lost the same course will be followed. But the fact that sometimes what is apparently, or even demonstrably, tuberculosis of the eye has healed spontaneously, renders such a course not altogether so plain, particularly where the diagnosis is somewhat doubtful. In this connection I will give the brief notes of a case which I considered to be tuberculosis of the iris, which was under my observation for some time and where the nodules completely disappeared, not, however, without having left almost total loss of vision.

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[1] Fano's (12) case is most probably one of inoculation by an operation for squint.

Harry H. was sixteen years old when I saw him for the first time at my clinic on May 22, 1885. At that time the following notes were made :

Tumor on the iris at the lower inner quadrant reaching up to the iris angle, length 2 *mm*, breadth 1 *mm;* its ant. wall lying up against the posterior surface of the cornea. Old iritis, with pupillary adhesions and deposits on ant. capsule of the lens. A leash of vessels on conj. running up to and slightly on the cornea, just over the location of the tumor. Simply p. l., in R. L E normal. There was a hard nodular tumor at upper outer edge of each orbit (enlarged lachrymal glands ?).

On September 29th, it is noted that there was a small yellowish nodule at the upper outer part of the pupillary edge of the iris similar to the one just described and enlargements of the pre-auricular and submaxillary glands were observed on the right side. Occasional attacks of pain and redness. January 12, 1886. Has been on inunctions of mercury for three weeks, but no ptyalism was produced. The swelling of the lachrymal glands diminished as are also the pre-auricular and submaxillary glands. July 16th. Not seen since May. In meantime has been under homœopathic treatment. R eye : almost total abolition of anterior chamber. Iris surface entirely free from nodules ; pupil small and closed; punctate opacities on cornea. At lower outer part of the iris is a white atrophic space pyramidal in shape, its base corresponding to the former position of the tubercular nodule. T—1. No pain, but it easily becomes red; V = p. l. L eye: pupil medium, but adherent all round; surface of iris studded with yellowish nodules. At the lower periphery of the iris, at the place occupied by a large nodule, the iris tissue is drawn towards the cornea, which at that point is opaque; punctate deposits on cornea near centre. Counts fingers at three metres. After this I did not see him again, but I learned some two years after, that he was still alive, but almost totally blind in the left eye. Before the left eye was affected I exhibited him before the Medical Society of D. of C., and he was examined by a number of gentlemen who pronounced his lungs free of deposit. His general appearance while not robust was not unhealthy.

In this case, when first seen, I think few surgeons would have had the temerity to enucleate, even if the consent of the patient could have been obtained, and yet the final results as regards vision could have been no worse from enucleation than they were by the *laissez-faire* plan, and the possibility of a general infection would certainly have been lessened.

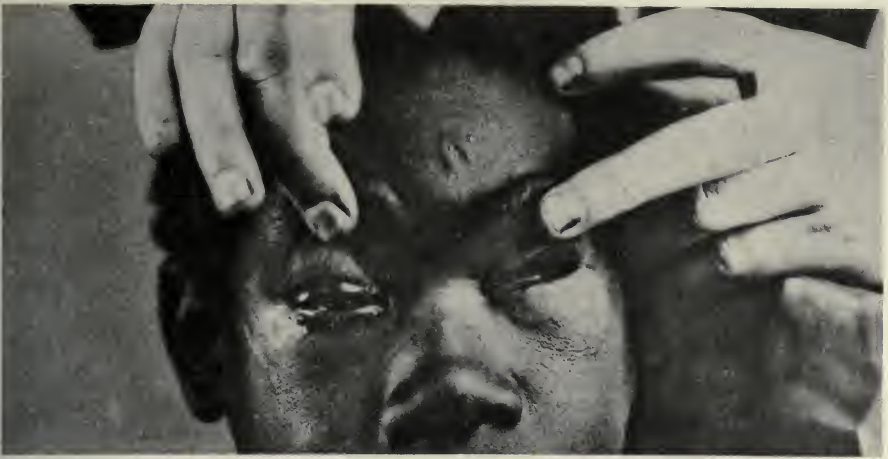
The diagnosis of the disease, microscopically and bacteriologically, is now a matter of certainty. The only diseases with which it is likely to be confounded clinically, are trachoma and epithelioma. The round yellowish nodules, with ulceration at points which mark its first stages, are sufficient to distinguish it from trachoma, particularly when taken in connection with the presence of enlarged preauricular and submaxillary glands which are considered by some excellent authorities, and it would seem with correctness, as constant accompaniments of tubercular affections of the eyes. Epithelioma could possibly be excluded by age, the majority of those affected with tuberculosis of the conjunctiva being young people. But the microscope will tell at once whether the diseased tissue is tubercular or not.

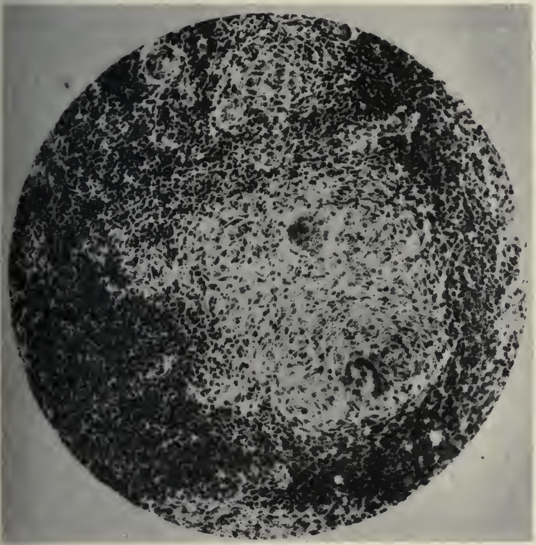
Explanation of Plate.

FIG. 1. Photograph from life, by Dr. Wm. Gray, of the Army Medical Museum, Washington.

FIG. 2. Photograph of a microscopical section (x $\frac{125}{1}$) of the excised conjunctiva. By Dr. Gray.

FIG. 1.





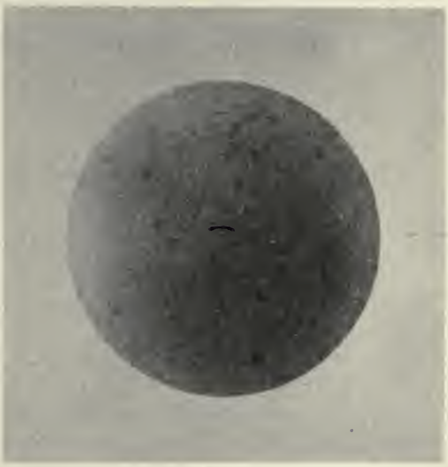
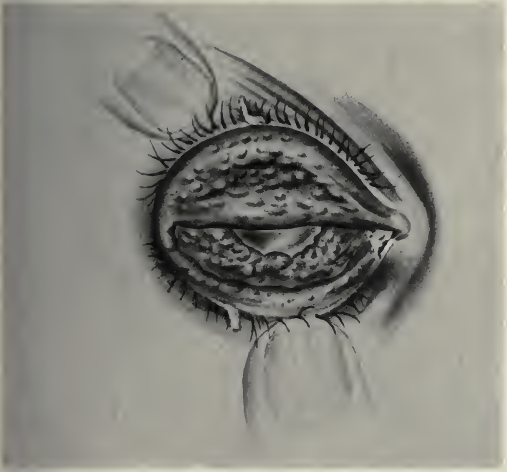


FIG.



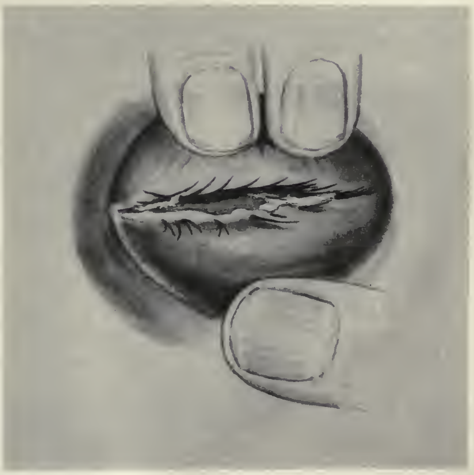


FIG. 5

FIG. 3. Photograph of a bacillus found in the scrapings of a diseased tissue (x $\frac{800}{1}$). By Dr. Gray.

FIG. 4. Everted lids of the right eye, showing nodules and ulcers of conjunctiva. From a colored drawing by Dr. H. V. Würdemann.

FIG. 5. Left eye showing the ankylo-symblepharon. From a drawing by Dr. Würdemann.

BIBLIOGRAPHY.

1. ABADIE. Tumeurs rares symétriques des paupières.—*Archiv. d'Opht.*, Juill.-Août, 1881.

2. ALEXANDER. Lupus der Conjunctiva.—*Zehend. Monatsbl.*, 1875.

3. AMIET, CASAR. Die Tuberculose der Conjunctiva. — Inaug. Disser., Zurich. Solothurn, 1887.

4. ARLT. Lupus der Conjunctiva.—*Zehender's Monatsbl.*, 1884, p. 332.

5. BAUMGARTEN. Ueber Lupus u. Tuberculose, besond. d. Conjunct.—*Virchow's Archiv*, 1880, also in *Gräfe's Archiv*, B. xxiv, 3.

5*a*. BENDA. Ueber die Beziehungen von Lupus vulgaris und Tuberculose. —*Deutsch. med. Wochenschr.*, 1886, No. 23.

6. BENSON. Primary lupus of the Conjunctiva.—*Dublin Jour. Med. Sci.*, 83, lxxv.; also in *Trans. Acad. Med. Ireland*, 1883.

7. BENSON. Lupus of the Conjunctiva.—*Oph. Review*, 1885; also *Trans. Oph. Soc. United Kingdom*, 1885.

7*a*. BRAILEY, W. A. Peculiar thickening of the anterior part of the conjunctival and subconjunctival tissue in a child, probably tubercular.—*Trans. Oph. Soc. U. Kingdom*, vol. ix, 1888–89.

8. CSAPODI STEFAN. Tuberculosis der Conjunctiva.—*Pest. med. chirurg. Presse*, 1889, No. 31.

9. COLLICA-ACCORDINO. Un cas de tuberculose de la conjunctive.—*Movimento*, Napoli, 1879, 2d Series, To. 1.

10. ELSCHNIG. Ein Fall von Tuberculose der Bindehaut.—*Wiener med. Presse*, 1889, No. 49.

11. FALCHI. Granuloma e tuberculosi di congiuntiva.—*Gior. d. r. Acad. Torino*, 1883, 3d Series, xxxi.

12. FANO. Tuberculose aguë de la conjonctive.—*Jnl. d'Ocul. et d'Chirurg.*, 1881, T. 9, p. 54.

13. FUCHS. Tuberculose der Conjunctiva.—*Wiener med. Presse*, 1886, No. 16.

14. GAYET. De la tuberculose conjunctivale.—*Bull. et Mem. Soc. Franç. d'Oph.*, 1885.

15. GERIN-ROZE.—*Soc. Méd. des Hôp.*, Feb. 10, 1882.

16. GRIFFITH, A. H. Case of primary lupus of the conjunctiva.—*Med. Chron.*, Manchester, xi, Novr., 1889.

17. HAAB. Die Tuberculose des Auges.—*Gräfe's Archiv*, xxv, 1879, Ab. 4.

18. HASNER. *Klin. Vortrage* ü. *Augenheilke.*, 1865.

19. HERTER. Casuistische Beobachtungen—Tuberculose Geschwüre der Conjunctiva.—*Charité-Annalen*, 1879.

20. Hock. Ueber Tuberculose d. Conjunctiva.—*Zehend. Monatsbl.*, 1875.

21. Hirschberg. Ueber tuberculœse Entzuendungen des Auges.—*Trans. Internat. Med. Cong.* 1881, London, also *Centralbl. f. prak. Augenheilk.* ii, p. 459.

22. Knapp, H. A case of tuberculosis of the conjunctiva with remarks.—*Archives of Oph.*, xix, 1890, No. 1.

23. Koster.—*Centralbl. f. d. med. Wissenschaften*, 1874.

24. Luc, H. De la tuberculose de la conjunctive comparée au lupus de cette muqueuse.—Thèse de Paris, 1883.

25. Laskiewicz. Ueber Lupus der Conjunctiva und Cornea, etc.—*Allgem. Wiener med. Zeitung*, 1877.

26. Leidholt, L. A. H. Beiträge z. Casuistik der Augen-Tuberculose.—1889.

27. Lowenthal.—Inaug. Dis., 1889.

28. Mules, P. H. Tubercle of the eye and its appendages in relation to general infection.—*Oph. Review*, vol. iv., 1885.

29. v. Milligan. Ein Fall von Bindehaut-Tuberculose.—*Centralbl. f. prak. Aughknd.*, July, 1882.

30. Manfredi. Contribuzione clinica et anatomo-patologica alla tuberculosi oculare.—Modena, 1876.

31. Maren, Emil. Beiträge z. Lehre von der Augentuberculose. — Inaug. Disser., Strasburg, 1884.

32. Manz, W. Zwei Fälle von Tuberculose des menschlichen Auges.—*Zehend. Monatsbl.*, 1881, p. 3.

33. Neumann. Ueber primäre lupöse Erkrankung des Auges.—*Wiener med. Wochenschr.*, 1876.

34. Pagenstecher u. Pfeiffer. Lupus oder Tuberculose.—*Berlin klin. Wochenschr.*, 1883, B. xx.

35. Pflueger. Lupus der Conjunctiva.—*Bericht.* ü *der Univ. Augenklin. in Bern.*, 1878.

36. Parinaud. Tuberculose primitive de la conjonctive; valeur des inoculations expérimentales pratiquées dans la chambre anterieur.—*Gaz. heb. d. méd. Paris*, 1884, T. xxi.

37. Peck, E. S. Case of conjunctival lupus.—*Archives of Med.*, iii, 333.

38. Pfeiffer, A. Tuberkelbacillen in der lupös erkrankten Conjunctiva.—*Berl. klin. Wochenschr.*, 1883, B. xx.

39. Pfleuger. Lupus Conjunctivæ.—*Zehender's Monatsbl.*, 1876, p. 162.

40. Rhein, K. Folliculäre Erkrankung d. Bindehaut des Auges.—*Gräfe's Archiv*, 1888, Ob. 3.

41. Rampoldi. Granuloma ulcerato o tubercolosi di congiuntiva.—*Ann. d'Ottal.*, 1886, xv, p.60.

42. v. Reuss. Fall von Tuberculose der Conjunctiva.—*Anz. d. K. K. Gesell. d. Aerztl in Wien*, B. 87, 1883; also another paper in *Eder's Aerztl.*, *Berlin*, 1888, Wien, 1889.

43. Rhein. Ueber primäre Tuberculose der Conjunctiva.—*Münch. med. Wochenschr.*, 1886, xxxiii.

44. Sattler. Tuberkulose des Auges.—*Bericht Heidelberg. Cong.*, 1877.

45. Sattler, H. Primary lupus of the conjunctiva.—*Irish Med. Jnl.*, 1874.

46. STŒLTING. Ueber der Tuberculose der Conjunctiva.—*Gräfe's Archiv*, 1886, Ab. 3.

47. TROUSSEAU. Inoculation du lupus, etc.—*Ann. Dermat. et Syph.*, 1889, 2d S. 704 ; also in *Archiv. d'Opht.*, Nov.-Dec., 1889.

48. ULRICH, G. Nachweis der Tuberkelbacillen bei Conjunctivaltuberculose.—*Hirschberg's Centralbl.*, 1885, p. 359.

48*a*. VALUDE. Ueber Tuberculose des Auges.—*Bericht. Heidelb. Cong.*, 1887.

49. VIRCHOW.—*Die Krankhaften Geschwülste*, B. ii, 1867.

50. WAGENMANN, A. Beiträge zur Kenntniss der tuberculösen Erkrankungen des Sehorgans.—*Gräfe's Archiv*, xxxiv, Ab. 4.

51. WALB. Ueber Tuberculose d. Conjunctiva.—*Zehend. Monatsbl.*, 1875.

51*a*. WALB. Ueber Tuberculose des Auges.—*Zehender's Monatsbl.*, 1877, p. 285.

52. WEISS, LEOPOLD. Ueber das Vorkommen von Tubercle am Auge.—*Bericht. Heidelb. Cong.*, 1877.

A METRIC SYSTEM OF NUMBERING AND MEASURING PRISMS.

(*Concluded from page 75 of this volume.*)

By CHARLES F. PRENTICE, of New York.

THE RELATION OF THE PRISM-DIOPTRY TO THE LENS-DIOPTRY OF REFRACTION.

SOME of the advantages of the prism dioptry, as the unit of measure for the refraction of simple prisms, having been shown, and whereas prisms are frequently combined with spherical lenses, it is here proposed to further consider the relations of the prism-dioptry to such combinations, as well as to the equivalents which are to be obtained by a mere decentration of the spherical lenses themselves.

In the accompanying figure, 8, a lens is shown with its principal anterior and posterior foci *f* and *F*, equidistant from *O*, upon the optical axis *f O F*.

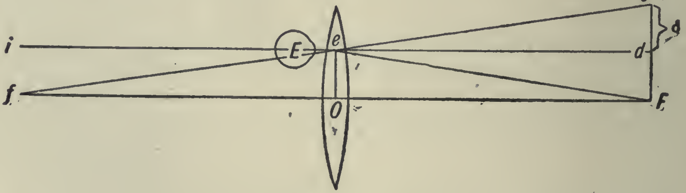


FIG. 8.

The ray, *ie*, which is parallel to the optical axis, and incident at an eccentric point *e*, of the lens, being refracted to the focal point *F* will sustain a deflection *dF*, *in the focal plane*, which will always be equal to the decentration *Oe*.

A ray, *de*, which is parallel to the optical axis and incident from the opposite direction, will be refracted to the focal point *f*, and, if received by the eye at *E*, will be projected by it in the prolongation of *fe* to *v*.

Consequently $dv = \delta$ will be the measure of the apparent displacement of the point *d*, resulting from the prismatic action inherent at *e*. By reference to the figure, and previous definitions, we then have

$$\begin{aligned} &\angle dev = \angle Ofe \\ &\angle deF = \angle OFe \\ \text{But } &\angle Ofe = \angle OFe \\ \therefore\ &\angle dev = \angle deF \\ \therefore\ &\delta = dF = Oe \end{aligned}$$

The tangent deflection δ, *at the focal plane of the lens*, is therefore always equal to the amount of decentration, and consequently in direct proportion to it.

Augmented decentration of the lens will be associated with an increase in the prismatic action, resulting from a growing inclination of the tangents $t_1\ t_2 \ldots$ determining the obliquity of the spherical surfaces, at corresponding opposite points of eccentricity, as shown in Figure 9.

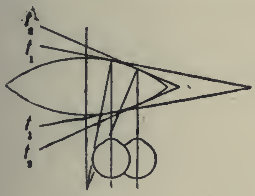


FIG. 9.

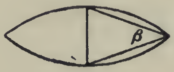


FIG. 10.

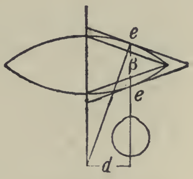


FIG. 11.

The inclination of these tangents, relatively to each other, becomes a maximum when the angle between them reaches 180°, that is to say, when they both coincide, and so form the tangent to the lens, which must then have become a perfect sphere. The amount of prismatic action it will be possible to obtain consequently only depends upon the diameter of the lens. It will be well, however, to bear in mind that lenses of large diameter, whose spherical aberration for peripheral rays causes the latter to fall short of the focus, will naturally also effect a still further increase in the prismatic action at the focus, for an extreme decentration.

It is further obvious that a *virtual* prism of *constant* angle cannot exist within the lens to produce the aforesaid variable results. The figure, 10, is therefore apt to prove misleading, as there is but one fixed amount of decentration, *d*, Figure 11, which corresponds to a prism of the angle β, and which is determined by the tangents to the lens surfaces at *e*, drawn parallel to the sides of the inscribed prism.

Thus prepared we may proceed to a consideration of the prism-dioptry.

Supposing a lens to be 1 D, and the deflection sought to be $1^{cm} = 1$ P-D, at the meter plane, which corresponds to the focus of the lens, it would require a decentration of one centimeter to produce this result.

A lens of 2 D, being decentred the same amount, would produce a tangent deflection equal to 1^{cm} *at its focus*, *half a meter*, which would be equivalent to 2^{cm} at the meter plane, or 2 P- D, it having been shown that the prismatic refraction is in the inverse proportion to the distance at which the unit deflection is produced. The following tabulation will serve to make this clear.

Lens.	Decentration.	Tang. Deflections at the Focus.	Tang. Deflections at the Meter Plane.
1 D	1^{cm}	1^{cm} at 1 meter	$1^{cm} = 1$ P- D
2 D	1^{cm}	1^{cm} at ½ meter	$2^{cm} = 2$ P- D
3 D	1^{cm}	1^{cm} at ⅓ meter	$3^{cm} = 3$ P- D

This table also exhibits the fact that any lens is capable of producing as many prism-dioptries as it possesses dioptries of lenticular refraction, *provided it is decentred one centimeter.*

On the other hand the prism-dioptries will increase or decrease as the decentration becomes greater or less. Thus:

Lens.	Decentration in Centimeters.			Decentration in Millimeters.		
	1 cm.	2 cm.		$1\ {}^{m}/_{m}$	$3\ {}^{m}/_{m}$	$6\ {}^{m}/_{m}$
0.25 D	0.25	0.5	Prism-Dioptries	0.025	0.075	0.15
0.5 D	0.5	1	“ “	0.05	0.15	0.3
0.75 D	0.75	1.5	“ “	0.075	0.225	0.45
1 D	1	2	“ “	0.1	0.3	0.6
2 D	2	4	“ “	0.2	0.6	1.2

A lens of 2 D, limited by its size to a decentration of $3\ ^{m}/_{m}$, will afford 0.6 P-D, while a lens of 1 D, capable of a decentration of $6\ ^{m}/_{m}$, will produce the same prismatic effect, as shown above. In other words, a lens of one half or one third the power will require to be decentred twice or three times as much to secure the same number of prism-dioptries.

This we find graphically demonstrated in the accompanying figures, in which the dimensions of decentration and lens curvatures are exaggerated to better serve our purpose.

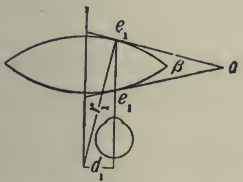


FIG. 12.

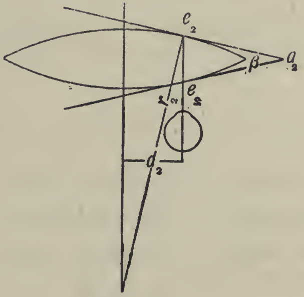


FIG. 13.

In Fig. 12 the lens of 2 D, with a decentration $d_1 = 3\ ^{m}/_{m}$, produces a deflection of 0.6 P-D *at the meter plane*, by reason of the obliquity of the lens surfaces, determined by the tangents at e_1 constituting a *virtual* prism of the angle β, with its apex at a_1.

In Fig. 13 the lens of 1 D, whose radius $r_2 = 2\ r_1$, is decentred by the amount $d_2 = 2\ d_1 = 6\ ^{m}/_{m}$, to produce 0.6 P-D *at the meter plane* by a virtual prism of exactly the same angle β, with its base at $e_2\ e_2$, and apex at a_2.

We have consequently but to remember that **the prism-dioptries in decentred lenses are in direct proportion to their refraction and decentration.**

Our present lenses will, however, not permit of a decentration of 1^{cm}, owing to their limited size, yet, if required, larger ones could be furnished the trade at a comparatively small increase in cost, which could be utilized specifically in corrections involving prism-dioptries.

However, in cases where the size of the lens proscribes the limits of decentration, it becomes necessary to add to it a constant prism. Since the slightest decentration of a simple lens is certain to produce a prismatic effect, it is evident that the *constant* value of any prism, which it may be desired to add to a lens, will only be retained when the optical axis of the lens strictly coincides with the visual axis, see Fig. 14.

The effect of decentration will naturally be to increase or diminish the prismatic action of the constant prism, which has been combined with the lens.

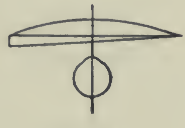


FIG. 14.

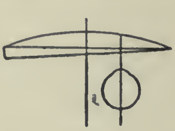


FIG. 15.

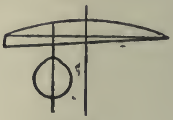


FIG. 16.

Thus in Fig. 15 the prism is increased by the prismatic action due to decentration of the lens, by shifting the visual axis toward the apex of the constant prism, while in Fig. 16 it is decreased by a decentration in the opposite direction.

Supposing a 5 D lens to be combined with a 2 D prism, the former being decentred 2 $^{m}/_{m}$, by shifting the visual axis toward the apex of the prism. We know that a 5 D lens will produce 5 P-D when decentred 1^{cm}, and therefore will produce 0.2 of 5 P-D when decentred 2 millimeters, which is equal to 1 P-D. The constant prism of 2 D has therefore been increased by 1 P.D, making it 3 P-D. A decentration of the lens to an equal amount in the opposite direction will leave but 1 P-D for the entire combination.

Two millimetres have in this case affected the value of the constant prism by 50 % of its active function.

We can now realize the importance and necessity of an accurate adaptation of spectacles, with regard to the pupillary distance, when high spherical corrections are resorted to.

The prism-dioptry and the meter-angle being directly dependent upon the pupillary distance, it behooves us, in any endeavor to secure accurate results, to be exceedingly particular as to its measurement.

Dr. Maddox, in his exhaustive work on prisms, calls attention to a unique, most simple, and accurate device for determining it.

Dr. Stevens having fully shown the disturbances occasioned by hyperphoria, I may here be permitted to call attention to the danger of artificially producing it by improperly centring the lenses in the vertical meridian in a simple case of hyperopia in which no real hyperphoria exists.

We shall admit that the lenses are decentred vertically, in opposite directions, by 5 $^{m}/_{m}$ above and below the horizontal plane, which will be equivalent to a decentration of one of the lenses by 1 centimeter, provided the other is properly centred.

Our hyperope being corrected by lenses of 2 D, would, under these circumstances, be forced to overcome 2 P-D, at the meter plane, and therefore a vertical diplopia of 12 centimeters at 6 meters, which amounts to 20 meters at one kilometer, or, approximately, in round numbers, 66 feet at a distance of 3,281 feet.

Inversely, in making Dr. Stevens' test[1] for hyperphoria, in emmetropia for instance, supposing a means to be devised to enable the patient to exactly indicate the distance between the images which he sees at a 6-meter distance. Admitting, by way of illustration, that he has decided them to be 6 centimeters apart, vertically, which, being equivalent to 1^{cm} at 1 meter distance, will lead us at once to decide that a prism of 1 D, properly placed before the eye, will correct his *manifest* hyperphoria. The same patient would have to struggle with a vertical diplopia amounting to $3\frac{1}{3}\,^{m}/_{m}$ at a reading distance of $33\frac{1}{3}^{cm}$, being quite sufficient to cause consecutive lines of type to appear intermingled with each other.

As a third demonstration, supposing it to be desired to afford binocular vision to an emmetrope at a distance of $\frac{1}{3}$ of a meter, *without his powers of accommodation or convergence being called into requisition.* His pupillary distance being 60 $^{m}/_{m}$, for instance, half of it will be 3 centimeters, which gives us 3 P-D for *his* meter-angle, making 9 P-D requisite to set aside his convergence to ⅓ of a meter, while 3 D of lenticular refraction are necessary to substitute accommodation for the same distance. A 3 D lens would require to be decentred 3 *cm* to afford 9 P-D, so that a pair of such lenses, placed before the eyes with their bases in, would accomplish the desired stereoscopic result. Two properly centred lenses of 3 D combined with 9 D prisms would serve the same purpose.

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[1] "Functional Nervous Diseases," by George T. Stevens, M.D., Ph.D., New York, 1887, page 194.

The above illustrations suffice to show the value of the prism-dioptry in leading to our conception of the actual work performed by prisms at different distances, and which the prevailing degree-system of numbering must continue to keep us in ignorance of.

Besides, a degree-system of numbering prisms cannot be brought to a *convenient* relation to any of the following points, which have been shown to exist in favor of the metric system :

1. A direct relation between the meter-angle and the prism-dioptry for variable pupillary distances.[1]
2. A direct relation of the prism-dioptry to the lenticular dioptry, for any amount of decentration of lenses.
3. Simple measurement of the pupillary distance determining the prism expressing the meter-angle.
4. All fractional intervals of the prism-dioptry being rendered available for differing pupillary distances.
5. The prism-dioptry being capable of measurement by a simple instrument, obviating it being taken for granted that the prisms have been correctly " marked."

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[1] This fully meets the suggestion of Dr. Maddox, who, in speaking of the decision of the committee of the American Ophthalmological Society, consisting of Drs. Edward Jackson, S. M. Burnett, and Henry D. Noyes, that all ophthalmological prisms should be *marked* with the angle by which they deflect rays of light, in his work entitled " Ophthalmological Prisms," on p. 30, says : " If I may be allowed to suggest it, a still better plan would be to have all prisms marked with meter-angles and their fractions, so as to correspond with lenses in the trial case, a meter-angle being the chosen unit of convergence, just as a dioptry is that of accommodation. The only disadvantage is that the meter-angle is an inconstant quantity."

It has, however, been satisfactorily shown that this seeming objection is an advantage to the new system, since the present inconstancy of our prisms can be utilized, thereby securing a degree of accuracy unattainable by any other means.

6. The resultant deflection produced by similarly placed superimposed prisms being equal to their sum expressed in dioptries.

7. Can be applied to existing commercial prisms, without increasing the cost or revolutionizing the present market.

The latter is certainly a most commendable feature, since any attempt to introduce prisms of special glass, or such that produce definite angular deviations, will heighten the cost, while depreciating the commercial value of those now on hand.

A possible objection to the new system of measuring might arise in the fact that it does not define the minimum deviation, yet, as the prisms used in spectacles are of small angles, " the difference is so trifling that it may be neglected in ophthalmic practice "[1]; and, as long as it is understood that prisms of greater angle are to be held with one side parallel with the base-line, from the eye, which is the case in measuring by the instrument, we at least obtain the desired uniformity.

In the event of it being desired to determine the resultant prismatic action of prisms which have been combined at any angle of crossing, we have but to resort to the statical formula:

$$R = \sqrt{P^2 + Q^2 + 2\,P\,Q \cos \gamma},$$

where P and Q represent the prisms, expressed in prism-dioptries, and γ the angle between their base-apex lines.

In ophthalmic practice this is apt to prove of possible value only when the prisms cross each other at right angles, consequently when $\gamma = 90°$, and therefore,

$$R = \sqrt{P^2 + Q^2}$$

The resultant prismatic refraction, R, will be in the plane which coincides with the diagonal of the parallelogram obtained by the forces P and Q.

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[1] " Ophthalmological Prisms," by Ernest E. Maddox, M. B., London, England, 1889, page 11.

CASE OF CORNEAL TRANSPLANTATION FROM THE RABBIT'S TO THE HUMAN EYE; RESULT NOT VERY FAVORABLE.

By WILLIAM F. SMITH, of Chicago.

(*With two figures.*)

December 6, 1888, Mary Katus, a Polish woman, was brought to my office by her brother. She is twenty-seven years old, very delicate, and slightly built. When she was three years old she suffered from an attack of small-pox, which left her totally blind.

The bulb of the left eye is soft and partially atrophied. The corneal membrane is entirely changed into cicatricial tissue. There is no perception of light. The sightless ball diverges and is in constant motion.

The right eye is nearly normal in size and tension. A dense leucoma occupies almost the entire extent of cornea, leaving only a very narrow margin (less than 1 *mm* in width) of partially transparent membrane just within the sclero-corneal junction. There is no visible anterior chamber, the iris apparently being firmly united to the posterior surface of the leucoma. The tension is not quite normal, making it probable that the lens was evacuated when the cornea was ruptured. Light perception, however, is good and the phosphenes are easily excited.

According to Von Hipple's rules it did not seem a case for which a transplantation could be done successfully. The relatives were very persistent, however, and I finally consented to make the attempt.

Von Hipple's instrument not being available, I had one made by Messrs. Sharp & Smith, of this city—a simple trephine without spring or clock work. It consists of a cutting-cylinder 4 *mm* in diameter and 10 *mm* long. This is cup-shaped and perforated through its bottom, the perforation furnished with a screw thread. Into this is screwed a shaft, 2 inches long, furnished with a milled head. Cutting-cylinder with shaft attached is shown at *A* Fig. 1. Over the shaft fits a metal cylinder just long enough to play

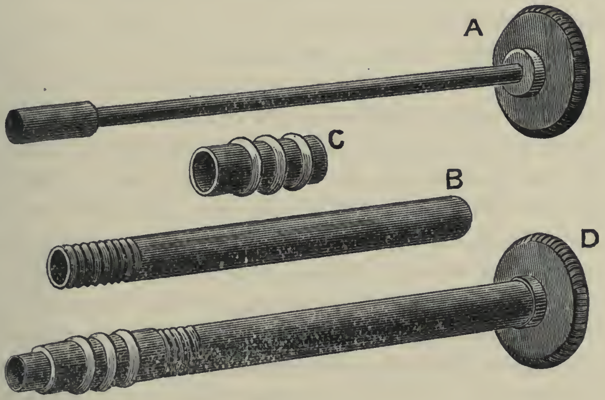


FIG. 1.

freely between the end of the cutting-cylinder and the milled head of the shaft. One end of this cylinder is furnished with a screw thread for the hood, *C*, which moves over the cutting-cylinder. Thg instrument when put together for use is represented at *D*. By means of the hood the depth of the incision may be exactly graded. The free end of the hood is square-cut. Its inner edge plays as closely as possible to the outer surface of the cutting-cylinder.

The patient was assigned a bed in the Emergency Hospital. A healthy rabbit, one year old, was procured. The operation was made December 13th. The most thorough antiseptic precautions were taken before, during, and after the operation. I was assisted by Dr. Hall and Mr. Schwann. Anæsthesia by chloroform was carried to as complete a degree as safety would allow. The patient proved a most excellent subject for chloroform, and took it kindly, with no struggle or any tendency to vomit.

The hood of my little instrument was fixed so as to expose exactly 0.9 *mm* of the cutting-cylinder. The point chosen for its action was about 2 *mm* downwards and inwards from the centre of the cornea. The instrument was held firmly with the left hand, while the milled head of the shaft was turned slowly with the right, so that the cutting edge, which was as sharp as it could be made, sank readily and evenly into the cicatrized cornea. The button to be removed was fixed by the fixation bident, with points reversed, and drawn upwards while it was carefully cut out with a small Graefe knife. This I found to be a very delicate piece of work, but was reasonably successful. The bottom of the mortise thus made was still opaque, except in one small spot not larger than half of the area of a pin's head, and it was impossible to determine whether or not this was a remnant of the membrane of Descemet. Further efforts to clear away tissue were abandoned for fear of penetrating the bulb. There was some little bleeding from the margins of the cut, which was stilled by a few drops of a solution of boric acid. The rabbit was now slightly narcotized with chloroform, and a few drops of a 10 % solution of cocaine were allowed to fall on its cornea. The nictitating membrane, or third lid, was cut across with a strong pair of scissors, as was also the orbicularis at the external canthus. This was done to prevent interference by spasmodic action on the part of these muscles.

The hood over the cutting-cylinder was moved back so as to expose several millimetres of the free end, and an attempt was made to cut through the clear cornea at one turn of the cylinder. This failed. The division was not complete, and as it was impossible to replace the knife so that it would complete the incision, the other eye of the rabbit had to be prepared. This was done after some little delay, when the second attempt, during which several turns of the cylinder were made, was entirely successful. The button of the rabbit's cornea had been forced up into the cup of the cutting-cylinder. The instrument was taken apart, and after the shaft had been removed the cutting-cylinder was fitted into the mortise already prepared in the patient's eye, and the button of cornea from the rabbit was forced down into it by means of a wooden tooth-pick fashioned for the purpose. The transplanted membrane lodged snugly in the place designed for it. It will be remarked that its surfaces had not been handled or in any way interfered with by any harsh instrument after it had left its natural location. The eyelid was drawn down over it and held by the hand of an assistant while the patient was, as gently as possible, brought from under the influence of the chloroform. The fact before mentioned, that there had been no vomiting, was of great assistance in carrying out the details of the operation.

The importance of remaining perfectly still was impressed upon the patient. She was carried to her bed, and I think I can say she did not move for twenty-four hours ; a trained nurse was at her side during all that time, and so assured me. To this fact, and to the gentle, tractable nature of the patient is due the perfect union that occurred, and was maintained till the healing process was complete. No bandage or packing was used other than a simple strip of gauze fastened round the head in such a manner as to hold the lids gently closed. I should have stated that the patient's face and eye had been cleaned with a boiled, saturated solution of boric acid, and with sterilized water. None of the usual irritating, antiseptic solutions had been used about the eye operated on.

For seven days the patient was kept, for the most part, in a recumbent position, after which she was allowed some liberty. The healing process seemed to be complete in about two weeks, and in three weeks after the operation she returned to her home in Streator, Ill.

At the time of leaving the hospital the transplanted piece was slightly elevated above the surrounding tissues, but the margin of union was smooth and presented no evidence of irritation. The button of rabbit's cornea had retained its transparency, but rested upon an opaque surface except at the small spot before mentioned, which was dark, and through which evidently some light reached the fundus. She could see any object held between the eye and the light, but could tell nothing about outlines. More could not be expected from one who had been so long totally blind.

About six months after the operation I was informed by the relatives of the girl that she could do "ordinary house-work"; and a few weeks ago a paragraph appeared in a local paper to the same effect. I had repeatedly written for the parents to bring the girl to the city in order to be able to report upon the condition after so many months. For some reason they were not able to come till last week, when her brother again brought her to the office.

Her general health has very much improved. She is now robust and stout. The transplanted piece of cornea has sunk to a level with the surrounding tissues, indeed somewhat below them especially at the centre, so that it appears like a facet polished on the surface of a sphere. The tissue upon which it has grown is not any more transparent than before. The small spot still has the dark appearance that indicates partial transparence, and she can now tell something about the outlines of objects held between her and the light. Her brother says she can tell the "chickens from the dogs and cats and other animals in the farm-yard," and that "she does considerable work about the house." The accompanying drawing, Fig. 2, is a rough sketch of the eye as it now appears to me.

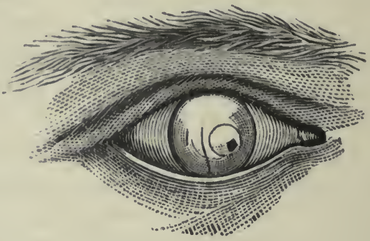


FIG. 2.

The line separating the transplanted piece from the rest of the cornea is not quite as well marked as here represented, and I have failed to show the facet-like appearance spoken of. The curved line passing from the centre of the cornea downward is a blood-vessel. The spot to the left of it indicates the location of a chalk-like deposit (calcareous matter) in the thickness of the leucoma. On examining closely the transplanted portion with a lens, it is easily recognized as transparent, and the bottom of the mortise can be seen as a rough surface dotted over by very fine points of the same chalk-like deposit. In the lower, inner quadrant is seen the dark, semi-transparent portion before described.

Both the patient and her brother say that her vision is improving. For some reason the very narrow margin of cornea described as existing at the first examination near the sclero-corneal junction seems to have widened, and the blue of the underlying iris is more perceptible. Perhaps her improved general health, and consequently increased activity constitute the reasons her relatives have for believing that her vision is actually improving.

I have attempted corneal transplantation five times in hospital practice, and this is the only case approaching success. It must be acknowledged that in it the circumstances were exceptionably favorable to successful union. The result cannot be considered very favorable. It only shows that in a case of central leucoma the cicatricial tissue of which does not include the membrane of Descemet, and where an anterior chamber still exists, a transplantation may be successful, and the transparency of the transplanted section may be maintained. But in just such a case an iridectomy would be a far preferable operation.

CHICAGO, March 26, 1890.

A SINGULAR CASE OF INJURY—BLINDNESS FROM A PENETRATING WOUND OF CHEEK AND ORBIT.

By WILLIAM F. SMITH, of Chicago.

January 2d, of this year, I was called in consultation with Drs. Lee and Murphy to see a boy who had become blind in his left eye from an accident.

The patient, twelve years old, healthy and well developed for his age, gave the following account of the injury. Himself and a few companions had been playing in a stable near his father's house. They were in a hay-loft, and he had accidentally fallen through a concealed opening to the floor below. One of his companions had been chasing him about the loft, and had in his hand a hay-fork with two tines. This fork fell, or was thrown, through the opening at the moment our patient fell, and when he reached the floor one of the tines of the fork went into his cheek. The fall in itself did not hurt him ; he was not bruised or dazed, but pulled the tine out of his cheek himself. When asked how far it had penetrated, he held up his hands so as to indicate about two and a half or three inches. He started to the house with his little friends. When he got to the door-step, he says he noticed that he was blind in the left eye. He was sure about this, because he said he had accidentally closed the right eye and found that he could see nothing at all with the other.

The mother said the boy had complained of very little pain, but being very much alarmed she had sent for the nearest doctor, who had simply put a few drops of an eye-water in the eye, the purpose of which she did not know.

The accident had happened forty-eight hours before I saw him.

Upon examining the eye we found that the pupil was dilated, but as the physician who had been first called had evidently used a solution of atropine, this was of no significance. The most careful tests failed to show that there was any perception of light in this eye. The ophthalmoscope showed the optic mediæ perfectly clear and the circulation of the fundus normal.

On the left cheek, about half an inch below the alveolar process of the superior maxillary bone and about half way between the chin and the angle of the lower jaw, was a small penetrating wound. It could be traced upwards, backwards, and inwards entirely through the cheek. Opposite the wound in the inner side of the cheek another opening, evidently the continuation of the wound, was found in the mucous membrane over the alveolar process and near the root of the second molar tooth. There was no discharge from this opening, and we did not penetrate it to any depth. There was no evidence of fracture anywhere, yet we had to conclude that the wound was in some way responsible for the blindness. The patient showed no evidence of any inflammatory process, there was no temperature, no headache, and no symptoms of cerebral lesion.

Upon returning to the office I took a skull to examine the relation of the parts. With a letter-opener, which is a long, narrow blade, I was surprised to see that when I pressed its point against the alveolar process of the second molar in the direction of the wound we had been examining, it found its way directly through the spheno-maxillary fissure at about its centre, across the apex of the orbit, and exactly bisected the optic foramen, the point being arrested by the upper anterior wall of that foramen. Indeed, pressing the point of the instrument anywhere within half an inch of the spot first chosen, the bones of the neighborhood formed a sort of funnel, which drove the instrument directly to the optic foramen. I was therefore forced to the conclusion that the tine of the fork had actually crushed the optic nerve just where it enters the orbit.

I have since made three examinations of the patient at intervals of ten days. At the last occasion I was able to determine that the optic disc had lost some of its color. The *arteria centralis retinæ*, however, seemed as prominent as ever, and the rest of the fundus had undergone no change. The eye became considerably diverged, and was displaced upwards. The pupil was dilated to a medium degree when the other eye was covered, but reacted promptly with the other. There has never been the slightest light-perception since the accident. I shall keep the eye under observation with considerable interest.

Has any *confrère* ever heard of a similar case ?

RELIEF FROM A GRAVE TYPE OF EPILEPSY FOLLOWING THE CORRECTION OF AN EXAGGERATED CONVERGENT STRABISMUS.

By CHAS H. BEARD, M.D.,

OCULIST AND AURIST NORTH STAR DISPENSARY, ASSISTANT SURGEON ILLINOIS CHARITABLE EYE AND EAR INFIRMARY, CHICAGO.

Peter Moos, aged fifty-two, a barber, a native of Germany, came for consultation to the eye and ear department of the North Star Dispensary early in March, 1888. The cause of his seeking advice was excruciating pain in the left eye, and affecting the sensory nerves over most all of the contiguous side of the face and head. This pain, which had its incipience in the eye, had been first felt some eight or ten years previous to his coming, and, recurring after shorter and shorter intervals, had grown to be almost constant. At about the same time had begun deviation inwards of the left eye, the pain and the convergence gradually increasing, one keeping pace with the other, until the one had become all but unbearable and the other had progressed to the extreme limit. It was only by a decided effort at abduction that a portion of the cornea could be brought into view. Four or five years previously the man had begun to be visited by paroxysms of an epileptic nature. These, too, had gradually increased in severity, while the intervals between the attacks had progressively shortened, till, at the time of his coming to the dispensary, their violence had become most distressing, and their frequency had attained an unwonted degree—as many as three or four, within a period of twenty-four hours, not being unusual. According to the description of them, given not only by the patient himself, but by several members of his immediate family, these paroxysms had every essential characteristic of ordinary epilepsy: falling down in total unconsciousness, convulsive movements, turning purple in the face, stertorous breathing, frothing at the mouth, etc. There seemed, however, to be no diminution in the power of the mental faculties. Indeed the patient was a man intellectually much above the average of his class, possessing a fund of practical knowledge, besides being well up in mathematics, astronomy, and the allied sciences, and, withal, an inventor of no mean repute.

It may be well to add that there was also in this case total deafness, evidently due to disease of the labyrinth, the later dating back for its beginning some twenty-five years. Aside from the abnormalities mentioned, the patient enjoyed excellent health. The morbid eye, from having once had acute vision, had grown quite amblyopic, the retina retaining its function, and that but feebly, on the nasal side only ; that is to say, objects, when held close up, could be indistinctly seen in the extreme temporal field. In all respects not mentioned, the eye was normal. Vision in the fellow eye was good, and there was no error in the refraction of either eye.

The pain about the eye was described as of a peculiar nature, chiefly a cruel pulling and straining backwards referred mainly to the side nearest the nose, and then an acute ache, extending over that side of the face and head.

The measure which seemed most obviously advisable was an operation for correction of the strabismus. The result could only be salutary if considered on no other than merely cosmetic grounds, and there was, moreover, a possibility of causing thereby amelioration of some of the more grievous symptoms. Accordingly, on the following day the patient presented himself fasting, and with the kindly assistance of Doctors Hall and Brougham, physicians to the dispensary, he was put under ether, and the operation was done. The anæsthetic was taken very badly, by the way, the man becoming, at one time, alarmingly asphyctic. We made an advancement, with a tendon resection, of the left external rectus, combined with pretty thorough tenotomy of the left internal rectus. Recovery was prompt, and the result most gratifying ; for not only was there correction of the deformity, but from that time till the patient's death, which occurred from a railway accident on the 22d June, 1889, fully fifteen months later, he neither suffered from the old agonizing pain, *nor had a single fit of epilepsy.*

A significant feature of this case is to be found in the fact that, immediately preceding death, there was again a slight tendency of the left eye inwards, and with it suspicions of pain and other uncomfortable symptoms, identical in character with those which had accompanied the former progressive convergence, began manifesting themselves. And, although nothing of an epileptic nature was ever again experienced, the morbific signs were, to the patient, so unmistakable as to create within him the keenest anxiety. I do not doubt, therefore, but that had he lived a year or two longer another, and a similar, operation would have been required.

The strabismus occurring thus late in life, the pain which bore a direct ratio to the aggravation of the squint, both having succeeded central ear disease; add to this the very happy result of the operation, and lastly the perverse tendency to secondary deviation of the globe, with its attendant symptoms,—do not all point to some disease, or source of irritation, at the base of the brain, the same causing decided lack of balance in the muscular system of the left eye, this, in turn, giving rise to reflex epilepsy?

CORTICAL HEMIANOPSIA WITH SECONDARY DEGENERATION OF THE OPTIC NERVE.

BY H. SCHMIDT-RIMPLER, PROF. IN MARBURG.

(*With plate ix. of vol. xix., Germ. Ed., 1889, and two illustrations in the text.*)

Translated by Dr. F. H. PRITCHARD and ALBERT BICK, of Boston.

THE subjoined case is one of unusual interest, it being, as it were, an experiment upon man in confirmation of Munk's investigations as to the seat of the centre of vision in the occipital lobe, furthermore in giving us information as to the position of the nerve fibres in the optic nerve which supply the hemianopic part of the retina.

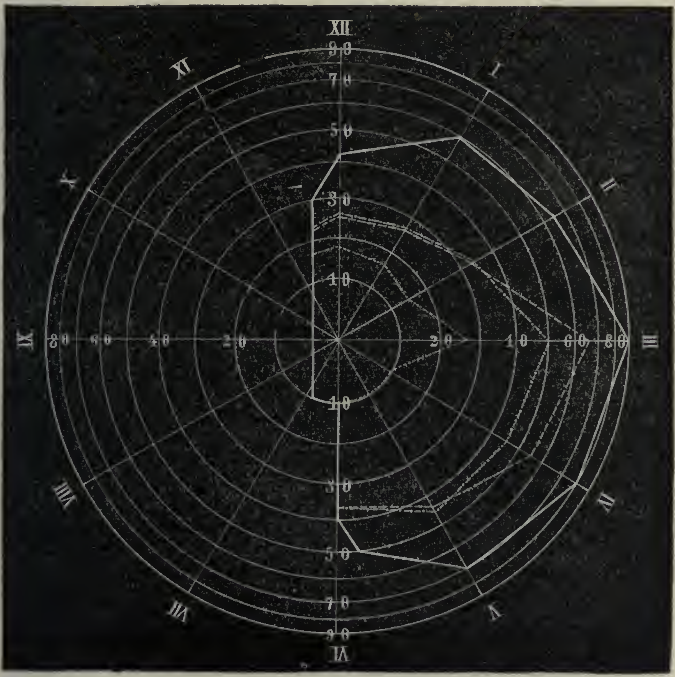
The patient in question, first received in 1882 into the surgical clinic of Prof. W. Roser, and observed by me for five years as to his sight, died in 1887, in the clinic of Prof. Mannkopff, of tuberculosis pulmonum. Already in 1882, in the Marburg Medical Society, a short report of his case was given by W. Roser and myself (*cf. Berliner klin. Wochenschr.*, 1883, 32); in the dissertation by Gerhartz (Marburg, 1885), a complete history is to be found, where especially is its remarkable course surgically considered. Prof. Marchand has also given, in the Marburg Med. Society, August 1, 1888, a detailed macroscopic description of the brain of this patient. The microscopic examination will be the subject of a dissertation to appear later.

H. Muhm, thirty-three years of age, received February 22, 1882, from a blow with a spade a comminuted fracture of the posterior portion of the **right parietal bone** a few centimetres below the sagittal suture. At the bottom of the wound in the skin, which was 5 *cm* long, running from in front backwards, with smooth, gaping edges, several splinters directed deeply inwards were visible. On the 23d of February the skin wound, in which there was was brain-substance, was enlarged and several splinters of bone were removed with the chisel. The dura mater was torn in several directions in a spot the size of a one-cent piece. For five days there was fever and icterus, followed by a normal condition. April 7th the wound had closed with the exception of two small and profusely suppurating fistulæ, and the patient could leave his bed. April 21st he became worse again ; sopor. The fistulous wound was enlarged, a cerebral abscess opened, and about a tablespoonful of pus removed, whereupon consciousness returned. The protruding fungus was finally removed with the scissors. May 4th, there was an aggravation with coma, accompanied with paralysis of the left arm and leg. Up to May 18th the cerebral prolapse increased in size ; it was pencilled daily with chloride of zinc. Pieces of brain substance, the thickness of one's finger, were several times taken away. From the 19th of May the paralysis of the left arm and leg decreased, and finally disappeared with the exception of a feeling of weakness in the left arm. In the course of the next week the wound covered itself with skin. The patient had observed, according to his own statement, as soon as he regained consciousness, a disturbance of the sight of the right eye, which he noticed the more *as he had already lost the sight of the left eye in his ninth year in consequence of an injury.* A large adherent leucoma was the result, and the eye was amaurotic.

An examination in the surgical clinic of Dr. C. Roser revealed a **left-sided hemianopsia of the right eye.**

I examined the patient July 14th, and measured the field of vision of the right eye with Scherk's perimeter. A nasal hemianopsia was found. The line of separation did not pass perpendicularly through the macula, but, as is more often observed, it extended about 5°–8° beyond the point of fixation, and in the upper part of the field of vision also it projected somewhat beyond the vertical. The temporal part of the field of vision had normal boundaries ; also for colors. At the first examination of the field of vision the area sensitive to green seemed somewhat contracted, but later was found to be of normal size, so that this variation may be ascribed to inaccurate statements of the patient, a difficulty experienced to a certain degree even in accurate observers. In the course of the following years a series of perimetric experiments was made. Aside from the variations just mentioned, the extent of the visual field remained the same, even in the examination made a short time before his death, as the following illustrations show:

Right Eye.
Field of Vision: July 14, 1882. June 2, 1884.

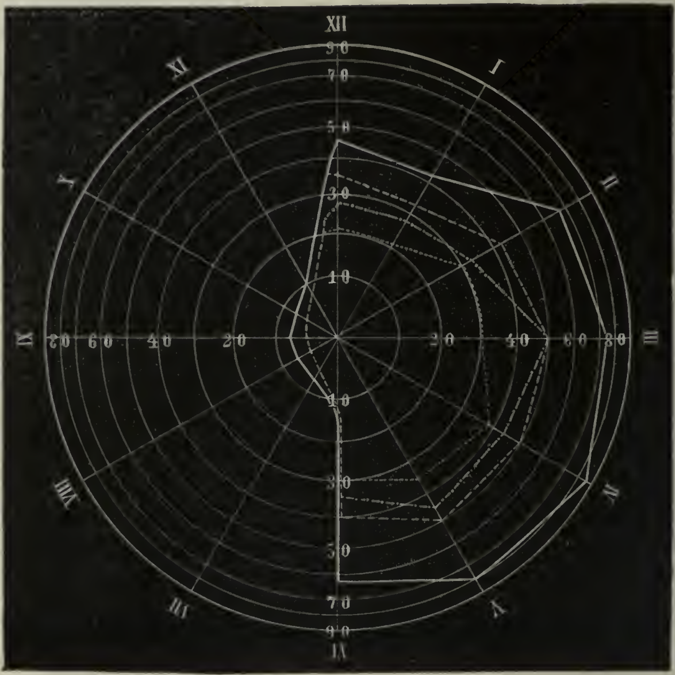


——— white.
---- blue.
—. —. — red.
.... green.

FIG. I.

The central visual acuteness of the emmetropic eye was normal and remained so. The P. proximum lay at six inches. The sense of light, examined with Förster's photometer, showed no disturbance. Externally the eye was normal; the ophthalmoscope also revealed no pathological changes. The optic papilla, as to color and amount of blood, was apparently normal; the temporal half appeared somewhat paler; yet this cannot with certainty be regarded as morbid, the pale color corresponding to the site of the physiological excavation. In the course of time a small part of the lower border seemed to have become somewhat paler than formerly. The pupil was of moderate size and responded to light. Alternate shading and illuminating of the hemianopic part of the retina yielded varying and uncertain results as to the reaction of the pupil.

Right Eye.
Field of Vision: Feb. 15, 1886 (examined with a white ball).



Feb. 15, 1887 (examination with colors).
Aug. 25, 1887 (slight deviations from that of February).
FIG. 2.

In the latter years, however, a greater difference was perceptible in the contraction of the pupil when light was thrown with the ophthalmoscope upon the nasal or temporal side; in the former the reaction was less marked, but yet it could be seen.

The patient, after the wound had healed, resumed his occupation and remained free from any paralysis or central disturbances. Yet sometimes his head would "get warm," and he would then be obliged to sit down; he did not have any real attacks of faintness or spasms. His memory is said to have become somewhat weaker. Sensation in the left hand was reduced. Hearing was good ; with his left ear he heard the watch not as well—at about half a metre—as with the right, there being a moderate catarrh of the middle ear. In February, 1887, symptoms of **pulmonary phthisis** showed themselves, to which he succumbed on the 17th of September (of the same year) in the medical clinic. At the **post-mortem** examination the brain was removed with the piece of bone attached to the cranial wound, so that it could be hardened *in toto.* The left optic nerve was flat, much thinned, and discolored on section ; the condition in question was clearly that of a centripetal atrophy originating in the left amaurotic eye. On section of the right optic nerve at the optic foramen a difference was distinctly visible between the color of the intensely white marginal part and the more grayish-white trunk. The left tract was much thinner than the right.

After complete hardening in Müller's solution and alcohol the right cerebral hemisphere, after previously sawing through the piece attached, was cut through in the direction of the sagittal suture about 3 *cm* from the median fissure. According to Marchand's communication, the section presented the following conditions : "The bone defect, situated in the posterior part of the right parietal, is 4 *cm* long. It is closed by a firm cicatrix, which is closely united with the brain to the same extent : the dura mater is lacking in this region. The right hemisphere is 3 *cm* shorter than the left, in consequence of a great diminution in size of the occipital lobe. The anterior border of the adhesion lies about 2 *cm* behind the interparietal fissure, the apex of the occipital lobe extending 1½ *cm* posteriorly beyond the place of adhesion. In the entire region of the latter the cerebral mass of the convexity is totally absent. The posterior horn of the right lateral ventricle is greatly distended (1½ *cm* broad), and in the region of the defect is distorted upwards, its inner surface covered with several folds. The space between the ependyma and the outer cicatrix was occupied by thick and quite fibrous cicatricial tissue, which also extended over the medullary substance of the apex of the occipital lobe. Within it there was found, between the floor of the posterior horn and the gray cortex of the under surface, a smooth cyst about the size of a hazel-nut ; this cyst was the product of softening. Only a very small part of the medullary substance in the outermost point of the occipital lobe was still to be found ; also the gray cortex to the same extent in the portion not yet affected of the lobe. Anteriorly, atrophic remains of some convolutions could still be recognized, embedded, however, in cicatricial tissue, the medullary substance being completely gray and sclerosed.

The medullary surface of the occipital lobe was partly adherent to the dura mater, and hence cannot be seen freely, but apparently it was sunken and shrivelled ; it was in its full extent down to its lower border shrivelled, while on the external circumference of the convex surface the convolutions were still intact.

The right optic nerve, hardened in Müller's solution and alcohol, was divided into sections from the optic foramen to the eyeball ; only a very small piece of the nerve lying near the optic foramen was used to make a longitudinal section. Differences in color could be seen, even in transverse sections of the nerve taken from the fluid used in hardening, as brighter and darker portions were distinguishable. The former were shown to be atrophic by Weigert's method of staining, which was principally employed ; specimens were also prepared with nigrosine, carmine, and hæmatoxyline. The normal bundles of nerve fibres appeared blackish or dark blue respectively upon using Weigert's method, and were of the usual size ; the atrophic ones were lighter, while more or less scattered darker rings were found in a yellowish tissue lying between ; they were much smaller in size. The connective tissue lying between the bundles of nerve fibres, which was colored brown, showed in the atrophied portions a narrower network, without broadening any of the septa. In longitudinal sections larger globular deposits, with a blackish contour, were found in the atrophic nerve-fibre bundles. Staining with hematoxyline revealed the free nuclei in larger numbers in the atrophic portions of the nerves ; but this may be explained by the disappearance of the nerve substance and the closer approximation of the nuclei. The position of the atrophic nerve fibres supplying the temporal half of the retina, which, if one accept semi-decussation, correspond to the uncrossed fibres of the tract, was varying ; in the vicinity of the optic foramen they appeared as a narrow seam, which surrounded about one half of the periphery of the nerves the middle of this crescent pointing inwards and downwards, *i. e.*, towards the nasal side. The temporal point extended only to the lower part of the temporal border of the nerve, while the nasal side, on the contrary, was but little removed from the uppermost point of the periphery of the nerve (*cf.* Plate ix., Fig. 1). Nearer to the eyeball a change took place (*cf.* Plate ix., Fig 2) ; in the vicinity of the point of entrance of the artery a wedge of healthy nerve substance penetrated into the inner lower atrophied periphery of the optic nerve (*cf.* Plate ix., Fig. 3). On coming nearer to the eyeball this part became broader (*cf.* Plate ix., Fig. 4), so that at the point where the vein enters, a broad transverse band of normal substance passes directly through the optic nerve, bounded above and below by a peripheric crescent of atrophic substance (*cf.* Plate ix., Fig. 5). This position of the peripheric crescents, both of which encroached somewhat upon the temporal side, was also apparent close to the eyeball ; the temporal portion of what had remained healthy of the nerve was larger than the nasal (*cf.* Plate ix., Fig. 6). The region of the lamina cribrosa was worthy of notice, inasmuch as the medullated nerve fibres first disappeared in the centre ; finally two wedges of dark-colored nerve bundles remained, a larger one on the temporal, and a somewhat smaller one on the nasal side, while at the periphery only a few dark dots could be found (*cf.* Plate ix., Fig. 7).

This case is an addition to the larger number of observations which support the view of Munk as to the seat of the centre of vision in the upper part of the cortex of the occipital lobe ; but scarcely any other shows with such clearness the traumatic destruction and its consequences. The injury affected the right occipital lobe, in its upper part, and soon after the left-sided hemianopsia was diagnosticated. At the post-mortem examination, only the outer extremity and the temporal convolutions of the occipital lobe were found unattacked by the suppurative processes following the injury. The nasal hemianopsia of the right eye—unfortunately the left one had been amaurotic since childhood—remained unchanged, with complete central visual acuteness during the following five years, up to the death of the patient. The secondary degeneration, an extension of which into the optic nerve Monakow and Siemerling have also recently observed in cerebral processes, extended through the optic nerve to the eyeball. It gives us certain information as to the position of the nerve fibres supplying the temporal half of the retina. They occupy in the vicinity of the optic foramen the whole lower periphery, a large part of the nasal and only the lowest part of the temporal side of the optic nerve. After the entrance of the vessels they lie more on the upper and lower periphery, extending especially from above and upon the temporal side; the nerve fibres supplying the nasal portion of the retina push themselves in between. If we compare herewith the position of the nerve fibres supplying the region of the macula as ascertained by Samelsohn, Vossius, Bunge, and others, which nerve fibres in the vicinity of the optic foramen lie in the centre of the nerve, but after the entrance of the vessels form a wedge with its base towards the temporal periphery, which position they maintain near the eye, we shall come to the following conclusions in regard to the distribution of the nerve fibres in the optic nerve. In the vicinity of the eye the fibres supplying the macula lie in a wedge-shaped bundle on the temporal side of the nerve, the fibres of the temporal portion on the upper and lower periphery encroaching somewhat upon the temporal as well as the nasal side, the fibres of the nasal portion in a part embracing the centre of the optic nerve and the middle third of the nasal half of the periphery in the neighborhood of the optic foramen, the fibres, which supply the temporal portion of the retina, occupy the lower periphery of the nerve, a larger portion of the nasal and a small lower one of the temporal periphery; those supplying the nasal portion of the retina occupy especially the upper periphery, a larger part of the temporal and a smaller upper one of the nasal periphery, whilst the macular fibres are found more in the centre.

As regards the position of the uncrossed bundle of fibres, as it was found in our case, the results obtained thus far are not uniform. But in the communications of Uhthoff[1], Jatzow[2], and Siemerling[3], which particularly come into consideration here, pure hemianopsia is not in question, so that the assertion that the uncrossed fibres should lie upon the *lateral* side of the nerve seems to me not to be proven; nevertheless individual differences are possible. It must also be considered that the secondary degeneration may be incomplete, and, further, that we cannot speak in a mathematical sense of an upper and lower part in the case of the optic nerve. In the case of Uhthoff, atrophy of the outer lower quadrant of the retina was found. The position of the fibres in our case agrees on the whole with that in Uhthoff's preparations (*cf.* Table V., Fig. 8). Even Siemerling, who states that the fibres of the uncrossed bundle in the trunk of the optic nerve lie laterally, reaching the periphery with the larger part of their circumference, in the orbital portion more below, shows in his Fig. 6 (beginning of the entrance of the central vessels) and Fig. 7 (about 5 *mm* beyond the entrance) no essential deviation from our results; the healthy uncrossed fibres lie principally below, although partially also in the centre. As in his case, besides the reduction of the central acuteness of vision, the greater part of lower nasal quadrant of the visual field had lost its perceptive power, as well as the outer half of the visual field, it will be easily understood that the portion of the optic nerve lying more to the nasal side, which according to my results belongs likewise to the uncrossed bundle of fibres, is shown not to be preserved in his transverse sections. The longitudinal sections, into which Siemerling has divided the optic nerve near the eyeball, do not seem, according to the cuts, to entirely confirm my results as regards the position of the uncrossed bundles. It is, however, difficult to form as exact a picture from the three longitudinal sections as can be done from the transverse sections, as the one passing through the upper part of the optic nerve still falls into the papilla, and therefore cannot show the upper periphery of the nerve. Finally, the fact should be once more emphasized that the case in question was not one of pure temporal hemianopsia, and that the central visual acuteness when last recorded, on November 21st, was only $\frac{15}{200}$. Death ensued on December 27th, and in the intervening time probably still other disturbances of vision took place. (On December 3d the record says: The patient opened her eyes a little, but apparently could not see the person addressing her.)

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[1] *Graefe's Arch.*, xxxii., Nos. 3 and 4.
[2] *Graefe's Arch.*, xxxi., No. 1.
[3] *Arch. f. Psychiatrie*, xix., No. 2.

The relation of the pupil to the action of light is also interesting. At the beginning the reaction was about the same, whether the percipient or the blind half of the retina was illumined. Later a difference in the extent and rapidity of the reaction of the pupil was noticeable, but the reaction was not entirely wanting, even when only a circumscribed portion of the blind half of the retina was illumined with the ophthalmoscope. The unavoidable diffusion of light comes here into play, and renders every such reaction unreliable. The explanation of this varying reaction of the pupil must be sought in the fact that, in the beginning, conduction in the optic nerve was unimpaired to the point at which the reflex in the corpora quadrigemina is transmitted, but later was destroyed in consequence of the secondary degeneration of the nerve-fibres.

Explanation of the Illustrations.

Figs. 1 and 6 were drawn by Dr. Weinbaum, the specimens being thirty-three times magnified. Fig. 1, of the size of the microscopic picture; Fig. 6, in a somewhat reduced size. In Figs. 2, 5, and 7 only the outlines and position of the healthy in contrast with the atrophic portions are shown; the former is shaded, the latter is left blank. Transverse sections are drawn, as seen from in front. The temporal portion is therefore to the left, the nasal to the right, of the observer. Fig. 1 represents a transverse section from the vicinity of the optic foramen, the following sections successively nearer to the globe. Fig. 7 corresponds to the vicinity of the lamina cribrosa, where already a large part of the healthy nerve-fibres appear unmedullated.

Fig. 1.

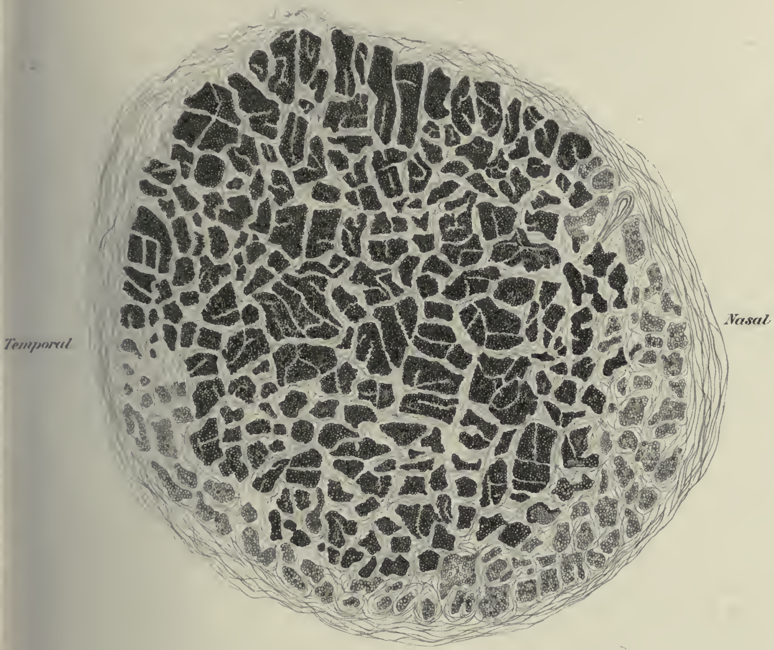


Fig. 2.

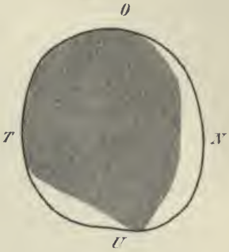


Fig. 3.

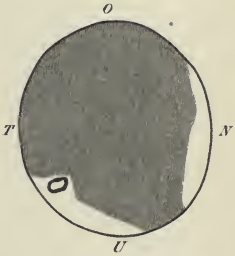


Fig. 4.

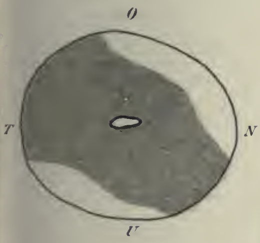


Fig. 7.

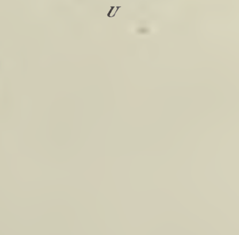


Fig. 5.

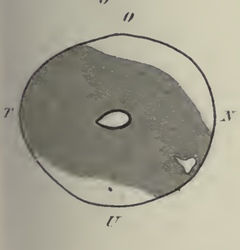
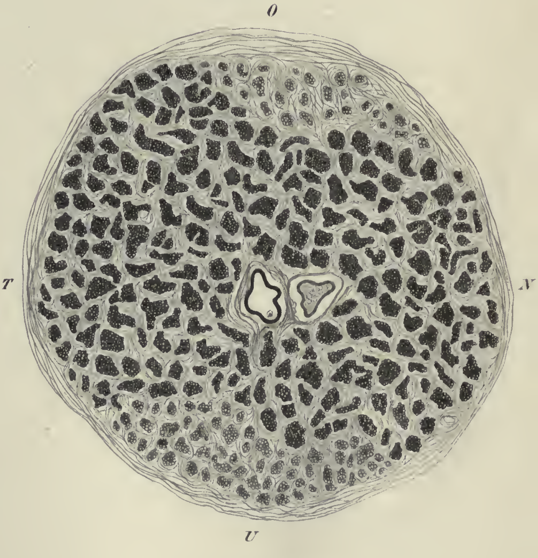


Fig. 6.



[illegible] Dr S Weinbaum

C Kirst, Leipzig

A CONTRIBUTION TO THE THEORY OF THE MUSCULAR RELATIONS OF THE EYES.

By D. H. SCHIÖTZ, of Christiania, Norway.

Translated by Dr. J. A. Spalding, Portland, Me.

(*With seven wood-cuts in the text.*)

WHEN the image of the object that we are gazing at falls simultaneously on the macula lutea of both eyes, we obtain simultaneous vision (fusion) with both eyes, because our central visual organ is so arranged that the images in the two eyes can only be comprehended as one. But in order that the image of an object at which we are looking can be depicted in the macula of both eyes, it is absolutely indispensable that the ocular muscles shall cause the visual lines of both eyes to intersect at the point upon which we are fixing our gaze. In other words, *the intersection of the visual lines must coincide with the point of fixation.*

After we have once attained the capacity of fusing the images of the two eyes, simultaneous binocular vision becomes an involuntary necessity, so that we stretch to their utmost extent the muscles of our eyes in order to maintain this simultaneous binocular vision whenever at a later period of life any thing interferes with the exercise of this function that has once become habitual and physiological.

Hence we see objects at a distance, as well as those near to, with both eyes together. The nearest point upon which, by the greatest possible exertion of the interni, we are able to converge the eyes, is the *near point of simultaneous binocu-*

lar vision (*fusion near point*). *Hence our far point for simultaneous vision with both eyes* (*fusion far point*)[1] *must be that point at which our visual lines intersect when we exert our externi to their utmost.*

We thus obtain *a range of simultaneous binocular vision* (amplitude or range of fusion) which embraces the space between the near and the far points of simultaneous vision (Nagel).[2]

In order to be able to investigate the muscular relations of the eyes, we must first know the precise position of these points.

The investigation of the fusion far point will of course coincide with an investigation of the abductive capacity of both eyes for distance and the method to be employed is the following: Whilst the patient observes an object (a candle or light, or as I prefer a vertical black stripe 2½ *cm* wide on a white background) at least 4 *m* distant, stronger and stronger prisms with the bases inward are brought before one of the eyes until the object appears double. The strongest prism with which the patient can still see the object single, and which he can "overcome" by diverging the eyes as much as possible, gives the angle between the visual lines in prismatic degrees.

The authorities differ in the strength of a prism that can be overcome by normal eyes, A. Graefe giving from 2°–5°, whilst Beselin[3] gives as high as 6°–8°. Since using a prismatic apparatus, which I will soon describe with a succession of prisms 2° apart, I have found that the maximum prism amounted to about 8°, and more rarely 10°, or 6° with the object at 4 *m*.

The *fusion far point* for normal eyes, therefore, equals a prism of 8° with *the base inward*, *i. e.*, the prism 8° with base inward is the strongest one that the divergence of the eyes can overcome in order to maintain simultaneous binocular vision, whilst the angle between the visual lines, presupposing that the position of the eyes was parallel, will measure 4° since the angle of deviation for the incident beam of light measures but one half of the angle of the prism, at least in the prisms that we employ.

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[1] We have left this expression in the translation, though the well-known term "binocular fixation" seems both simpler and clearer.—H. K.

[2] *Graefe-Saemisch*, Bd. vi., p. 489.

[3] THESE ARCHIVES, xiv., 200.

Our fusion far point in normal conditions is therefore a negative one; it lies about 1 *m* behind us.

Strictly speaking, some correction is necessary, because our visual lines are not parallel when viewing an object at 4 *m* distance, but they converge and form an angle of 53′, which really ought to be deducted from the above-mentioned angle of 4°, but practically speaking it need not be considered.

The fusion near point, or the nearest point to which we can converge whilst maintaining simultaneous vision, is simply determined by measuring how near to the eyes we can see a small object without perceiving double images. We use an illuminated point which is most easily obtained by holding toward the light a screen with a minute hole. I use the apparatus shown in Fig. 1; a thin black iron plate, a little larger than a playing card with a pin-head perforation in the middle. The aperture must be sharply defined as the cross-section *b* in Fig. 1 shows. Beneath the hole is a fixed tape divided into a centimetre scale.

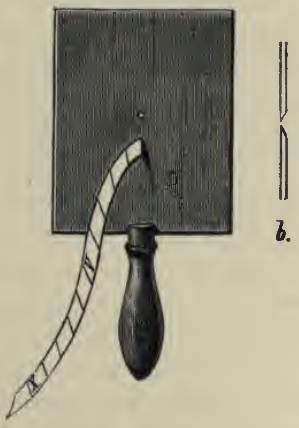

FIG. 1.

The patient stands in front of a bright window and holds the screen in front of him. As he fixes upon the point of light the screen is brought nearer and nearer, keeping the light point steadily in the middle point between the two eyes, until the former begins to appear double. By moving the screen a little closer or a little farther away, we easily find the point where the patient can maintain simultaneous binocular vision, and then we measure with the tape the distance between the screen and the external canthus. In this way we measure approximatively the one side of a triangle which is formed by the visual lines and the line between the rotation points of the two eyes, the base line, whereupon the angle of convergence, *i. e.*, the angle between the two visual lines can be reckoned for any given base line.

Landolt's dynamometer, constructed for the same purpose, has a metallic hat, with a minute hole in one side, that can be placed over a flame and then turned till the light shines through. Below is a metrical tape for measuring the angular distance.

The fusion near point for normal eyes is about 6 *cm*, when measured in this way, but as the distance between the eyes is about 6 *cm*, we have a convergence of about 60° between the visual lines.

If we find a fusion far point about 1 *m* behind us, and a fusion near point 6 *cm* in front of us, we get a range of fusion embracing not only the space in front of us extending from infinity up to 6 *cm*, but also partially the space behind us, that is to say from infinity to about 1 *m*. We call the former range our positive, the latter our negative range of fusion, or the amplitude of convergence and the amplitude of divergence.

Just as we speak of an amplitude of accommodation and transform it into dioptries, so can we speak of an amplitude of fusion and express it in degrees. Our entire range of fusion amounts to about 63°, about 60° for the space before us, and 3° for the space behind us.

In addition to the degree, we have another larger unit, which we can often practically employ as a measurement in our investigations.

Nagel (*Graefe-Saemisch*, Bd. iv., p. 478) suggested the idea of using the metre-angle as a unity, *i. e.*, "the fusion-angle for an object at a distance of one metre." The angle of fusion in this case is the angle which the visual line forms with its original position when the eye has rotated inward in order to arrange itself at the distance of 1 *m* from the object which lies in the middle line between the two eyes, that is to say, the angle "a" in Fig. 2.

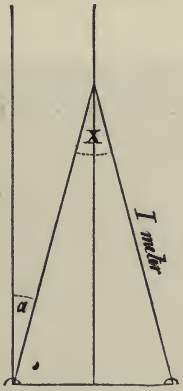

FIG. 2.

When, however, we speak of simultaneous vision, we have to consider the movements of both eyes, therefore, as I think *the angle of convergence between the visual lines of both eyes* for an object at a distance of one metre, is to be styled the metre-angle—that is, the angle $x = 2a$.

An angle of two metres will consequently be the angle of convergence between the visual lines for an object $\frac{1}{2}$ *m* distant, etc., etc.

The size of the metre-angle depends upon the distance of the rotation points of the eyes. Nagel averages this distance in adults at 64 *mm.* But since our investigations often include younger persons, I have ventured to take it at 61 *mm.* With this value, the metre-angle—*a*—will be 1° 45′, whilst the metre-angle *x*, *i.e.*, the angle of convergence between the visual lines for an object one metre off will be $3\frac{1}{2}°$, which is convenient for reckoning.

A prism of 7° will therefore coincide with a metre-angle. If prisms could only be ground according to the metre-angle system, it would be of great practical value, and mechanically I do not suppose that there would be any difficulties in the way.

Fig. 3 exhibits the curves for the metre-angles.

Our range for simultaneous vision will measure about 18 metre-angles, 1 for our negative, and 17 for our positive range of simultaneous vision.

With normal muscular relations, however, our visual lines are not only compelled to intersect in the fixation point, if we are to see with both eyes, *but the same thing must happen if one eye is excluded from the visual act.*

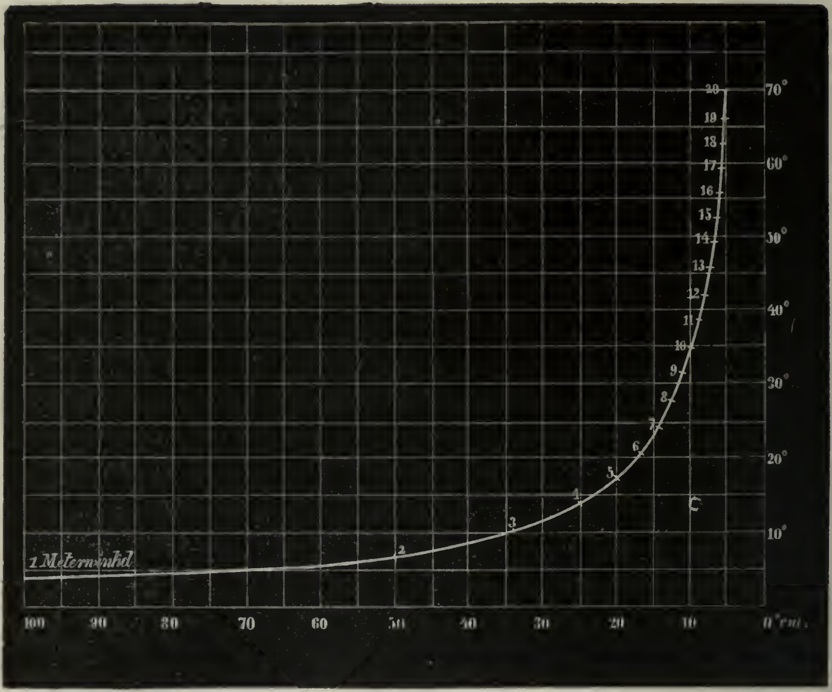

FIG. 3.

If an object is fixed, and one eye is covered with the hand, the latter is obliged to remain in its position behind the covering hand, neither moving inward nor outward; *equilibrium must exist for the object-distance.*

A preliminary investigation of the relations of equilibrium is easiest made with the covering hand. We let the patient fix an object at the distance for which we wish to investigate the equilibrium, and we cover one eye, *e.g.*, the left eye, with the hand. On removing the hand and covering the right eye, we observe whether the left eye remains quietly in its fixation position, and then we either find equilibrium, or a movement of the eye outward or inward, in order to arrange itself anew upon the object, which proves of course, that the eye has not maintained its position behind the hand, but when excluded from the visual act it deviated inward or outward, and so far as the object distance is concerned has converged or diverged. Having observed how the left eye behaved, we again cover it and watch the right eye, a procedure which can be many times repeated.

If equilibrium is not present we must next discover whether simultaneous vision is still intact.

The patient fixes the object as before and covers one eye with the hand and then removes it, without then covering the other eye, and now we examine whether the eye at once arranges itself for simultaneous vision, or whether it remains in its incorrect position and as it were delays to arrange itself. The simultaneous vision is then in greater or less degree "*dissolved.*" Since only one eye shows this sluggishness in arranging itself, we must investigate both eyes in the same manner.

Equilibrium exists for all distances from infinity to 10 to 12 *cm*, provided that the muscular relations are normal. We therefore obtain an *equilibrium far point* in infinity, *an equilibrium near point* at a distance of from 10 to 12 *cm*, and *a range* (*amplitude*) *of equilibrium.* (Mesoropter. H. K.)

The equilibrium far point lies somewhat within the far point for simultaneous vision, the equilibrium near point slightly outside the near point for the same.

The visual lines when arranged for the equilibrium far point are parallel, when arranged for the equilibrium near point, they form a convergence angle of from 30° to 35° *i.e.*, 8½ to 10 metre-angles. The amplitude of equilibrium is somewhat smaller than that of simultaneous vision, and lies slightly within the limits of the latter.

A more accurate determination of the conditions of equilibrium is to be made by v. Graefe's test of holding a prism of at least 10° with its *base downward* before the left eye, *e.g.*, when we have double images which vary so much in height that they cannot be fused into one. But when binocular vision is abolished, the eyes arrange themselves according to the equilibrium between their muscles, and if this is normal, the visual lines will still intersect in the fixation point. If we now employ a flame, for distant testing and a black point on a white ground or an illuminated point for a near test, the patient will see two lights or two dots or points *directly above one another*, the upper one of which will belong to the left eye.

If, however, equilibrium for distant objects is lost, the double images will not lie directly over one another but *obliquely above one another*. If convergence is present in proportion to the distance of the object, we shall have homonymous images—the upper one, that of the left eye, will stand to the left and above the lower image, whilst in divergence, which produces crossed double images, the upper light will be above and to the right of the lower one.

If equilibrium is absent we must next discover how much the visual lines deviate from the position of equilibrium. The usual way is to bring in front of the eye (the right eye in this case) stronger and stronger prisms with the base outward in convergence, and with base inward in divergence, *until the images stand directly above one another*, when we shall have in prismatic degrees the convergence or divergence of the visual lines in proportion to the distance of the object.

This method is satisfactory for long distances, but when they are short, we get too great values, especially with divergence. And at all events the method is clumsy and takes much time.

If instead of a light or point we employ a horizontal ruler divided off into centimetres we shall see two rulers when we hold in front of the left eye a prism of 10° with its base downward, and if the eyes are equally balanced, the two rulers will stand one directly over the other. If convergence is present, the upper ruler will be pushed to the left; and if divergence prevails, to the right, in relation to the lower one. Then as the ruler is divided into centimetres we have only to note by how many centimetres the upper ruler is pushed one way or the other over the lower, when we have at once the direct deviation from a position of equilibrium expressed in degrees.

I have used this method for years, and found it as practical as it is convenient.

I generally examine the equilibrium for three distances; 3.45 *m* (5.75 *m* if the room is large enough), 1.15 *m*, and 0.286 *m*. We can now divide the ruler for distances of 3.45 and 1.15 *m* into whole or half degrees, or into metre-angles, or, as I have found most suitable, into centimetres, because I then can employ the ruler for both distances. My ruler is 1 *m* long and 7 *cm* wide, and can be hung over the back of a chair with hooks. It is white with black lines, for every 2 *cm*, so that every second stroke goes clear across the ruler.

It occasionally happens that the prism of 10° is not sufficient for 1.15 *m*, in which case we place a stronger prism in front of the eye, or an additional weak prism in front of 10°, till the difference in height is so great that there is an open space between both rulers.

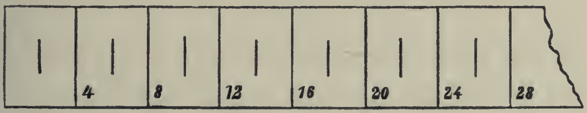

FIG. 4

There is no need of subdividing the ruler according to the tangent to the angle for so slight a distance as we need. Thus for 15° the arc of a circle with a radius of 3.45 *m* is 90 *cm*, and the tangent 92.4 *cm*, which is of very slight account in our investigations.

The ruler is to be placed in such a way that its *left* end stands directly in front of the patient, and is the one to be looked at. He must then say whether both ends of the two rulers that he sees stand directly one above the other, or whether the upper one is longer or shorter than the lower, *i.e.*, whether the upper one is pushed to the left or to the right in comparison with the latter, and if this is the case he must count off by how many long strokes on the ruler the upper one is longer or shorter. The dislocation in centimetres is then transposed into degrees.

There is generally but little difficulty in reading off the ruler. If the dislocation is extreme, and the patient is a child, counting off the strokes at the greater distance is somewhat difficult, but if we pass a cane vertically along the ruler to and fro, the patient can generally tell how much the lower ruler projects beyond the upper. For this reason in case of convergence, the right-hand end of the ruler is the one to be fixed.

For these reasons where the displacement is extensive, the examination ought not to be carried on at a distance farther than 1.15 *m*. The displacement at this distance is much less, and the legibility of the lines greater.

At 0.286 *m*, I use a thick horizontal stroke on a white card board, divided into centimetres.

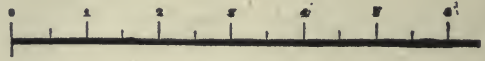

FIG. 5.

The figures above the strokes are about the size of Jaeger I., so that we can be sure that when the patient sees them distinctly his eyes are really arranged for the proper distance. In this test a displacement of 2 *cm* will correspond to 4°, one of 4 *cm* to 8°, etc. The line must be 10 to 12 *cm* long.

The tests for equilibrium are of the greatest importance, and these, in combination with the determination of the fusion far point and fusion near point, form in my opinion the only useful foundation for the determination of the muscular relations of the eyes.

I will now exhibit a short list of cases with normal muscular relations:

1) Student of medicine B. H.
+0.5 ; +0.5
$<\frac{20}{30}$ $<\frac{20}{20}$
5.75 metre 0
1.15 " 0
0.286 " 0
Fusion far point Pr. 8°
Fusion near point <6 *cm.*

2) Student, F.
" " " ; " " "
$\frac{20}{20}$ $\frac{20}{20}$
5.75 *m* 0
1.15 " 0
0.286 " . . . . Div. > 0
Fusion far point Pr. 8°
" near " 6 *cm.*

3) K., æt. 33.[1]
52° − 0.5 ; 145° − 0.5 − 5.5
3.45 *m* Conc. 3 *cm* = ½° with − 4.4
1.15 " Not measured.
0.286 " Conv. < 0: with − 3 ; − 3
Div. > 0: without lens.
Fusion far point Pr. 8°
" near " 6 *cm.*

4) Miss K., æt. 18.
0° − 1 − 6 ; 150° − 0.75 − 6.5
$<\frac{20}{20}$ $\frac{20}{30}$
3.45 *m* . . . . . 0 with − 5 ; − 5
1.15 " . . . . . . Not tested.
0.286 " . . . . . 0 without lens.
Fusion far point Pr. 8°
" near " not measured

5) A. B., æt. 16.
" " + 5 ; " " + 5.5
$>\frac{20}{50}$ $>\frac{20}{50}$

Has lenses + 4 : + 4 to read with : lately used constantly.

3.45 *m* Conv. 3 *cm* = ½°
1.15 " " 0
0.286 " " 0
Fusion far point Pr. 8°
" near " 11—12 *cm.*
} Without lens.

3.45 *m* Conv. 2 *cm.*
1.15 " " 0
0.286 " Div. > 0
Fusion far point Pr. 6°
" near " 12 *cm*
} With + 4: + 4

We can include amongst the cases with normal muscular relations not only those in which the equilibrium is absolutely normal, but those with an insignificant variation one way or the other, provided that they do not surpass one half a degree.

It would be superfluous to mention that by the mark < > we mean that the value discovered approximates the one given.

We must therefore arrange three formulæ according to the equilibrium for the distance, which I will call *the static position of equilibrium*, viz.:

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[1] For the explanation of these symbols see these ARCHIVES, xv., p. 203. It is the designation of the meridians according to Javal-Schiötz. The number with a degree is the deviation of the axis from the vertical meridian on the right side ; the next number is the strength of the cylinder in dioptries ; the third, the spherical glass in dioptries. The number below is the visual acuteness ; on the left of the " ; " is the left, on the right, the right eye ; the glass seen from behind.—H. K.

(1) The parallel equilibrium position.
(2) The divergent equilibrium position.
(3) The convergent equilibrium position.

The two latter conditions may offer difficulties. But even with parallelism for distance, we often see difficulties for near work, muscular asthenopia. Before entering upon this topic I will describe my prism apparatus, and mention the examination of the capacity for ab- and adduction.

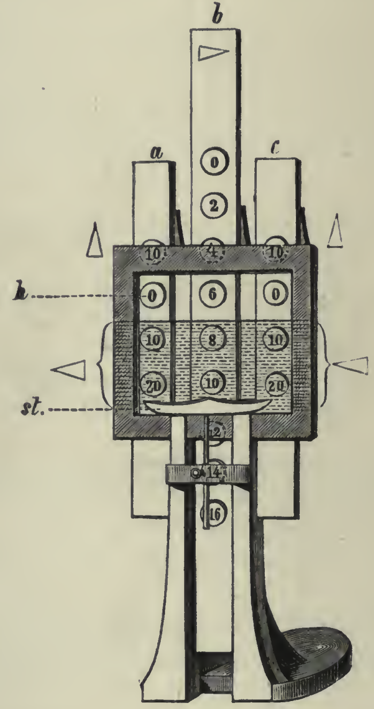

FIG. 6.

Fig. 6 shows the apparatus from behind. The three staves with prisms *a*, *b*, and *c*, can be pushed up and down, and remain fixed by means of a spring. The figures give the number of the prisms, and the triangular figures show the position. The eyes of the patient look through "*o*" "*o*" opposite "*h*." The horizontally shaded portion is closed with a thin plate in order to prevent chafing the glasses.

When the patient supports his chin upon the left side of the rest "*st*," so that he can look through the prisms in *a* and *b*, prisms as high as 36° with the base *in*, can be pushed in front of his eyes: 20° in front of the left eye and 16° in front of the right. If on the contrary he places his chin to the right, and looks through the prisms in *b* and *c*, prisms with their bases *outward* up to 36° can in turn be brought in front of his eyes: 16° in front of the left eye, and 20° in front of the right.

The equilibrium tests are begun by pushing the staves entirely down, so that the prism 10° with its base downward and the "*o*" opening are in the middle, so that the prism can be placed at pleasure before either the left eye or the right.

There is no need of wasting further words upon the apparatus; it is plain, that the examination must be more rapid and accurate when this is employed than when we are obliged to exchange and arrange each prism with our hands.

The investigations of the fusion far point and near point have given us a glance into the absolute capacity for ab- and adduction on the part of the eyes, or, as we used to say, the capacity for convergence and divergence.

It is also of great importance to fix the relative capacity for divergence and convergence for a definite distance, and we have consequently chosen 4 to 6 *m* for the longer distance, and 0.33 for the shorter, employing as a test a flame or a vertical black stripe on the wall; for near test we employ a black stripe on white paper.

After bringing stronger and stronger prisms before one or both eyes with the base inward for determining the divergence, and with the base out for determining the convergence, we find the strongest prism which the patient can overcome, still maintaining binocular vision.

If we wish to use Snellen V1 for 6 *m*, or Snellen 0.5 for 0.33 *m*, we find that the limit for distinct vision is far from coinciding with the limit for simultaneous binocular vision, at all events not in the convergence experiments.

We will next refer to the relation between convergence and accommodation.

Let us imagine that we have a young emmetrope with good accommodation; his range of accommodation will about coincide with the range of convergence, and for the same object-distance, he will accommodate about as many dioptries as he will converge in metre-angles. Thus, for example, with the object at 33 *cm*, he must accommodate 3 dioptries and converge 3 metre-angles.

Accommodation and convergence are, however, not generally so narrowly connected with one another. We find, particularly in prism experiments, that the same patient whilst maintaining his accommodation at 3 dioptries, *i.e.*, whilst still maintaining his distinct vision, can converge a great deal more than 3 metre-angles. He can converge up to 6 or 8 or even more metre-angles, and on the contrary he can reduce his convergence, that is to say, he can diverge in proportion to the distance of the object up to one half a metre-angle or even up to his fusion far point.

In the same way, as we shall find by experiments with concave and convex lenses, whilst maintaining his convergence of 3 metre-angles, he can accommodate up to 6 or 7 dioptries, and again relax his accommodation down to 1.5, or 1, or even to 0 dioptries.

We find, in other words, that for a definite arrangement of accommodation, there is a relative amplitude for simultaneous vision, the convergence amplitude of which amounts to from 3 to 5 metre-angles, and depends upon individual variations and the situation of the fixation point, and the divergence amplitude of which rotates somewhere about the 3 metre-angle.

If we test the capacity for convergence on the part of these emmetropes for 6 *m* with a lamp flame, we shall approach the limit of his relative convergence for 0 dioptries when we come to a prism of 15°, 20°, or 25°. Then must arise a struggle between the distinct, and the simultaneous binocular vision. If the patient cannot abandon distinct vision, he can no longer converge, and with stronger prisms double images will arise. In this case we thus discover his relative capacity of convergence for 0 dioptries of accommodation. To judge from the values that are given as normal for distances of 4 to 6 *m*, I feel justified in assuming that generally this relative capacity of convergence is fixed.

If, on the contrary, binocular vision conquers, the patient abandons distinctness of vision, and accommodates and converges more and more, and it will then depend chiefly upon practice (if the muscular relations are normal) how strong prisms he can overcome. Personally I have full control over my convergence, and I think that I can bring into play my entire capacity of convergence. I have never had at my command a combination of prisms higher than 90°, but this I can easily overcome.

This was the case with Em. But with Hm, or M, the relations between accommodation and convergence are different. In M the range of accommodation is pressed more or less toward the near point of the range of fusion; in Hm the range of accommodation is pushed out toward the fusion far point, and even partly beyond. In both, the convergence for a distance of 4 to 6 metres will stand in an entirely different relation to the accommodation, than in Em, and this must influence the result of the investigation. Yet any test which can be influenced as well by exercise as by the condition of the refraction, and a struggle between binocular vision and distinctness of vision, cannot have much if any value.

I carried on these tests for a long time, but found the results so varying, the gain so doubtful, and the time employed so enormous, that I abandoned them.

Alf. Graefe[1] gives the following values for the capacity of convergence and divergence:

Distance > 4 *m*.	Distance 33 *cm*.
Div. Pr. 2° — 5°.	Div. Pr. 5° — 10°.
Conv. Pr. 20° — 30°.	Conv. Pr. 30° and over.

Beselin[2] has found greater values for the divergence and smaller for the convergence, as well as Reich, whose results are noted by Beselin:

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[1] *Graefe–Saemisch* Bd. vi., p. 41.

[2] These ARCHIVES, vol. xiv., p. 219.

Beselin	Div. Pr. 6.2°.	Div. Pr. 11.5°.
	Conv. 13.2°.	Div. 20.2°.
Reich	Div. 5°.	Div. 15°.
	Conv. 15°.	Conv. 24°.

The capacity of divergence for distance coincides with the determination of the fusion far point, which corresponds generally to a prism of 8°.

I have discovered convergence for distance as high as 30°, and as low as 18°, 14°, 12°, with entirely normal muscular relations.

I have not tested so fully for the near point, but I have found the capacity for divergence from 7° to 16°, and for convergence from 16° up to 70°.

These extreme variations show that we have not yet found a stable foundation for determining the muscular relations.

It has, however, been asserted that the relation between divergence and convergence with normal muscular relations ought to be about $\frac{1}{2}$, but the figures given by A. Graefe do not show such a proportion, and, so far as my investigations go, there is some such proportion in some cases, but not in others. One circumstance, however, is often discovered, that when the proportion is greater than $\frac{1}{2}$, or even than $\frac{1}{1}$, an increased capacity for divergence or defective capacity for convergence will be present.

Great stress has been laid upon the importance of the accommodation for the convergence, but I think that this has been exaggerated.

If we have a Hm of 4 D, an Em, and a M of 4 D, we find that the Hm must use 6 D of accommodation for an object 50 *cm* away, the Em 2 D, and the M no accommodation for the 2 metre-angles of convergence, which are needed to arrange the eye for the object. The convergence angles, always constant for any fixed distance, are therefore united in various cases with variable quota of accommodation, which depends upon the refractive condition of the eyes. On the contrary, constant quota of accommodation must appertain to the determined angles of convergence in every individual case.

When, however, in the above examples we correct the Hm and M with the respective + and — 4 D lenses, and the proportion once existing between accommodation and convergence is suddenly deranged, what then occurs? The patient will feel uncomfortable for a short time, since he must employ for the same metre-angle of convergence a suddenly different quota of accommodation. For this reason he will at first have difficulties, be obliged to determine slight variations in distance, and must, *e.g.*, pass carefully up and down stairs, but these difficulties soon disappear since the accommodation and convergence quickly adapt themselves to the new relationship.

I will later speak of the influence which a sudden derangement of the relations will exercise upon the convergence when binocular vision is absent.

The necessity for simultaneous binocular vision compels us to converge correctly; by it we learn to converge a 2 metre-angle for an object at a distance of ½ a metre, and 5 metre-angles for an object ⅕ metre distant. And the accommodation? Well, that has to arrange itself accordingly. A person with Hm 4 D may learn how to use 6 D of accommodation for 2 metre-angles of convergence. If emmetropic, he may learn how to employ 2 D of accommodation, and with myopia of 4 D he has to learn how to converge for 4 metre-angles without any accommodation.

In order, however, that definite angles of convergence shall always belong to definite distances, "*a sensation of convergence*," as Alfred Graefe has entitled it, will develop itself, and so far as the accommodation is concerned, a so-called "sensation of accommodation" will develop itself according to the relations existing in the given case between the accommodation and the object distance.

To this sensation of accommodation we owe the disagreeable feelings which are perceived when the once arranged proportion between convergence and accommodation is suddenly deranged. For a given distance (convergence) an accommodation must now be employed which was formerly employed at a different distance, and this must influence our judgment of various distances until we have accustomed ourselves to a correct appreciation of the condition of affairs, *i.e.*, until the adaptation between the accommodation and convergence is again established.

I must be able to judge of the distance by means of the "sensation of convergence," but I cannot comprehend that with the same I could also regulate my convergence as some believe. The sensation of convergence cannot, of course, have any influence upon the *accommodation* convergence.

There is, however, one influence, which, in addition to the necessity for simultaneous vision, must play a certain rôle, and this is the "consciousness of nearness," as first suggested, so far as I recall, by Hansen Grut.[1]

I will take the liberty of calling it the "consciousness of distance," because I shall then embrace objects at every distance.

The consciousness of distance is developed by exercise and experience, generally under the associated action of convergence and accommodation, though we see also that it is influenced by both, but it must be regarded as chiefly independent of these. We observe particularly, that even the one-eyed have a consciousness of distance. Although they are obliged to dispense with the fine shades of difference at short distances (which belongs to binocular vision and by means of which Hering's gravity test alone is possible), yet they are never in doubt whether an object is one metre distant or only half that distance.

We must owe this particular consciousness of distance to our capacity of being able to judge the situation of an object in proportion to its surroundings, according to the mutual size, position, and distinctness of the retinal images, as well as from our knowledge based upon experience of the size and situation of the objects. And this consciousness must be of considerable importance for our convergence; at least we can easily prove by experiments some conditions which cannot otherwise easily be explained.

Thus, if I bring a convex glass of 1, 2, or 3 D before my eyes and thus make myself myopic (I am emmetropic) and then test the equilibrium for 0.286 *m* in the method before described, I find instead of equilibrium with + 1 D, a divergence of 2°: with + 2 D a divergence of 4°; and with + 3 D a divergence of 6°. With corresponding concave lenses, I find the same degrees of convergence.

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[1] Compte-rendu. Copenhagen, 1884. Latent squint, especially latent divergence.

When the once adapted proportion is suddenly deranged, and simultaneous vision is abolished, the influence of the accommodation upon the convergence is satisfactorily demonstrated.

More careful examination shows, however, a regulating influence which we nevertheless must owe to the consciousness of distance.

Without any glass I accommodate for a distance of 0.286 *m;* 3.5 D, and I converge 3.5 metre-angles or somewhat more than 12°. With + 1 D I accommodate 2.5 D or for a distance of 40 *cm*, but I converge 12° — 2° = 10°, or for a distance of 35 *cm*. With + 2 D I accommodate about 1.5 D, *i.e.*, for 67 *cm*, but I converge 12° — 4° = 8°, *i.e.*, for an object at a distance of 44 *cm*. With + 3 D I need but ½ D of accommodation, and nevertheless my eye is accommodated for an object 2 *m* distant. My visual lines, however, still form a convergence angle of 12° — 6° = 6°, and consequently I am converging for an object distance of 58 *cm*.

Hence we see that the convergence does not follow the changes in accommodation, but that the eyes invariably converge for a distance less than that of the accommodation employed.

In other tests I have also discovered conditions which we must owe to the "consciousness of distance."

I believed for a long time that the stereoscope could take the place of prisms in investigations concerning convergence and divergence. Thus if we know the distance between the glasses in the stereoscope and that between the objects, we can easily discover, by means of a curve, the convergence-angle of the visual lines. But we soon found that the results obtained with the stereoscope did not agree at times with those found with prisms; with the stereoscope, I found a slighter capacity for divergence and a greater capacity for convergence than with prisms.

It frequently happened, that patients with normal ocular muscles, with a fusion far point corresponding to a divergence of from 3° to 4° were unable in the stereoscope to arrange their visual lines parallel; the accommodation was of course abolished since the objects in this test stood at the foci of the lenses and the patient was Em. But he could not free himself from the consciousness that the object was close to the eyes, and for that reason was unable to put his accommodation completely at rest.

Just in the same way as Snellen's tables cannot be made good by the images in an optometer, just so little can stereoscopic pictures take the place of genuine objects at their natural distance in examinations of the muscular relations. In both cases the "consciousness of distance" will be led astray and thus the accommodation and the convergence will be influenced.

It is now time to take up the threads of a previous section of this paper, where we stopped at the subdivision of the static position of equilibrium into the following three groups:

1. The parallel position of equilibrium.
2. The divergent position of equilibrium.
3. The convergent position of equilibrium.

I mentioned before that under all of these conditions difficulties at near work were likely to ensue. These discomforts, which we call "muscular asthenopia," do not reveal themselves by such characteristic symptoms as in accommodative asthenopia, where the oppressive pain over the eyes and the rapid haziness of objects are constantly complained of.

Muscular asthenopia is generally characterized by a biting pain in the eyes when working at near objects as well as by lachrymation, and a feeling of weariness in the eyes which is often associated with pain radiating backward into the head and occiput. We rarely hear complaints of objects becoming indistinct, wavering, or double. And this must depend on the circumstance that the exertion to preserve the fusion produces the above-mentioned symptoms, which compel the patient to abandon his work before the muscles have totally surrendered their service.

Frequent squinting and nipping of the lids is a characteristic symptom occasionally encountered when the muscular relations are abnormal.

Since these symptoms, however, are observed not only with abnormal muscular relations, but may also, *e.g.*, be due to a blepharitis or conjunctivitis or to some nervous affection, the test with the covering hand should never be omitted.

We will now more closely investigate the especial forms and begin with the first group.

First Group.—Parallel position of equilibrium for distance.

To this group belong, first, all cases with normal muscular relations, where we find a fusion far point corresponding to a prism of 8° with base inward, the fusion near point about 6 *cm*, and equilibrium for the three distances.

To this group belong also cases in which we have a normal fusion far point, and parallel position for equilibrium, but *divergence for near.* Even at 1.15 *m* the patient asserts that the upper ruler stands somewhat to the right (prism with base downward for the left eye) corresponding to a divergence of 1° to 2°, and a still greater divergence, 2°—8°, will be noticed for the distance of 0.286 *m.* Under these circumstances, then, the equilibrium near point must be pushed farther away, as far as 2–3 *m*, or even to the equilibrium far point, so that often the amplitude of equilibrium is lost or is but very slight in extent.

If the conditions of equilibrium are such as these, the fusion near point must be pushed farther outward but to a proportionally less degree; we generally find it from 9 to 20 *cm* distant, rarely farther. Such a removal of the equilibrium near point and of the fusion near point can only be due to defective converging capacity, and upon what this latter depends we do not yet precisely know.

We have already mentioned the relation of accommodation to convergence, and that this is important is clear enough. The co-operation of accommodation and convergence is the reason why every exertion of accomodation involuntarily produces an inclination to convergence, and *vice versa.* In myopia, particularly when of high degree, the convergence is forced to dispense with some support, but this may be of no account since we have already seen, in the examples of normal muscular relations, that even a myopia of 6 D has not caused any disturbance of convergence.

It is my opinion that a developing myopia will only obtain an importance for the muscular relations, when the convergence is more or less defective from the start; under such circumstances the myopia might bring out defective capacity for convergence. In judging the importance of myopia, we must also consider how much more frequently it occurs in proportion to emmetropia and hypermetropia in those persons who use their sight a great deal for near objects.

A unilateral amblyopia will be of similar importance for the convergence, especially with an extreme variation between the sight of the eyes. In such a case, the fusion will be easily dissolved, and a defective convergence so much the easier come to light. The real cause will have to be sought, either in the muscles themselves, or in the condition of the innervation. The term, insufficiency of the interni, as we call this condition, shows that defective capacity for convergence has been chiefly regarded as a sequence of diminished muscular energy.

The interni work in two ways, partly with the externi in the associated movements of the eyes, partly alone, in preserving the convergence. With insufficiency we find no limitation of the movements of the eyes in lateral rotation, whilst a considerable defect of convergence may exist. If the lateral movements of the eyes demanded as much muscular energy as is demanded in the movements of convergence, we should be obliged to conclude that the defective convergence could not depend upon a weakness of the muscles, which perform the whole work in the associated movements; the insufficiency must, in this case, originate from anomalous innervation.

Nevertheless, we are scarcely justified in identifying the muscular work produced by two different motions.

We have found for normal eyes that the fusion near point lies at about 6 *cm* from the rotation point of the eyes. The angle of convergence between the visual lines will then be about 60°, therefore the rotation of each eye will amount to about 30°. According to Alfred Graefe,[1] the eyes in associated lateral motions can accomplish a version of from 42° to 45°.

We find, therefore, that the interni, when normal and working in the service of convergence, accomplish only about ⅔ of the motion which they are able to give to the eyes when they act associatedly with the externi, and yet (if we grant to the sensations which we perceive in carrying out this experiment, any importance at all), we feel distinctly that the interni are stretched and exerted differently when we converge, than when we use them in associated lateral movements. This signifies that the convergence makes greater demands upon the interni than do associated lateral motions, and it may suffice for an explanation to say that the lateral motions can still be accomplished with ease, whilst the convergence is still defective. We must also reflect that the eyes are only held for brief intervals in the extreme lateral positions, whilst the convergent position is the most customary.

The term "insufficiency of the interni" was not a happy one, because we cannot positively affirm that the muscles are actually at fault, and because the name has not only been employed in the condition of things with which we are now occupied (a parallel position of equilibrium for distance with defective convergence), but also in the cases which belong to the next group, divergent position of equilibrium for distance.

The most correct term would be "*convergence insufficiency*," which adequately expresses the condition of affairs without making any attempt at an explanation of the cause.

The following case is a remarkable example of convergence insufficiency:

Student, æt. 22. 90° — 0.75 — 1.5 ; 80° — 0.75 — 1.5
$\frac{20}{30}$ *p. p.* 10 *cm* $\frac{20}{30}$.
3.45 *m.* Conv. > 0.
1.15 " Div. 4 *cm* = 2° }
0.286 " " 3.5 " = 7° } Without lens.
1.5 " = > 3° with — 2 ; — 2.

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[1] *Graef-Saemisch*, Bd. vi., p. 11.

Fusion far-point prism 8°.

Fusion near point about 20 *cm;* the patient cannot state positively that he has got rid of the perception of double images ; but one eye invariably deviates outward when a point of light or a book is brought up to a point about 20 *cm* away.

He complains of burning and weariness in the eyes after reading a short time ; also of tension in the eyes, which he is all the time blinking and winking. He has used a lorgnette of 2 D concave for reading and can then use his eyes considerably longer.

Even when he converges to the maximum extent, the eye does not turn in any more than so as to leave a distance of about 2 *mm* between the inner corneal margin and the punctum lachrymale. In associated movements, however, the inner corneal margin turns way over to the caruncle.

Our patient has a positive range of fusion, or range of convergence of scarcely 5 metre-angles. There is scarcely any range of equilibrium, but there must be a point of equilibrium at a distance of about 3 meters.

The myopia is rather slight, but surely not without importance, not, however, as a cause, but because, as formerly mentioned, the convergence is thereby partly deprived of a support of which, in the preceding case, it stands so much in need.

The determination of the fusion near point in this case could only be carried out approximately, because the patient had got out of the habit of seeing crossed double images. As we witness this very rarely in pure convergence insufficiency, we shall describe it more in detail in the next section. It is peculiar that these individuals cannot abandon fusion, and that they let one eye follow its longing to deviate, when they cannot possibly grasp the crossed double images that arise during divergence. Long-continued practice is the only thing that can bring about this desired condition and the outcome of it all in the majority of cases is a manifest deviation for objects near at hand.

It is quite natural that in a case so well pronounced as this, asthenopia should appear, but the point of the greatest interest for us to discover is how great the deviation in the equilibrium experiments can be, and how far the fusion near point can move away before the asthenopia begins.

Without speaking decidedly, I think that I have discovered that when the divergence for the distance of 0.286 *m* is less than 3°, and when the fusion near point is less than 10 *cm*, asthenopia will scarcely be felt in pure convergence insufficiency.

We must bear in mind these two points: the less the fusion near point is removed, the better the deviation from equilibrium can be tolerated.

The dislocations of these points often stand in a suitable relation to one another, as we see from the following case:

7) Miss H. V. ″ ″ ″ ; ″ ″ ″
$< \frac{20}{20}$ $< \frac{20}{20}$
5.75 *m* 0
1.15 " . . . . Div. 3 *cm* = 1.5°
0.286 " . . . . " 3.5 " = 7°
Fusion far point, prism 7°. Fusion near point, 20 *cm*.

8) Miss K. ″ ″ ″ ; ″ ″ ″
$< \frac{20}{20}$ $< \frac{20}{20}$
3.45 *m* . . . . Div. 2 *cm* = 0.3°
1.15 " . . . . " 4 " = 2°
0.286 " . . . . " 1.5 " = 3°
Fusion far point, prism 8°. Fusion near point, 11 *cm*.

I believe, however, that the equilibrium deviation for 0.286 *m* is often proportionally greater than the removal of the fusion near point, and particularly in myopes, that we can find distinct divergence in the equilibrium tests united with an almost normal fusion near point.

9) Mr. A. 50° + 1.25 − 0.5 ; ″ ″ ″
5.75 *m* . . . . Div. 4 *cm* = 0.4°
1.15 " . . . . " 4 " = 2°
0.286 " . . . . " 3.5 " = 7°
Fusion far point, prism 9° ; near point, 11½ *cm*.
(He closes the left eye after reading a while, and especially by artificial light.)

10) Miss M. æt. 14 ″ ″ − 5 ; ″ ″ ″ − 6
3.45 *m* . . . . 0 *cm* with − 4 ; − 5
1.15 " . . . . Div. 0 " " − 3 ; − 4
0.286 " . . . . " 0.5 " " − 3 ; − 4
. . . . "0.25 " = 5° without lens.
Fusion far point, prism 6°; near point, 7 *cm*.

It may be advantageous to distinguish between these two groups—between those with a fusion near point, removed to a considerable distance and those in which this point is but slightly removed. The latter still have a large amplitude of fusion at their command, and they can be more easily assisted than the former, whose amplitude of fusion is noticeably diminished.

Landolt[1] has asserted that one must have an overplus of at least $\frac{2}{3}$ of his range of fusion in order to work for any length of time. When the work is held at 33 *cm*, the patient uses a convergence of 3 metre-angles; in order to endure this work, the patient must have at command a convergence of over 3 metre-angles. The entire positive amplitude of fusion would amount to 9 metre-angles, and the fusion near point lie at a distance of 11 *cm*.

Landolt grants, however, that he has often found that a still greater reserve fund is needed, even as high as $\frac{3}{4}$ of the amplitude of convergence, and as I have discovered, that the fusion near point ought not to lie farther off than 10 *cm*, we probably need more than $\frac{2}{3}$ of the amplitude of convergence as a reserve fund, but for the sake of brevity we can use this fraction in the calculation.

If we have a patient with a fusion near point of 14 *cm*, and consequently with a positive amplitude of fusion of 7 metre-angles, he will at a working distance of 33 *cm* have 2 metre-angles too little. Fortunately it will not be the question of compensating for these metre-angles, but only $\frac{1}{3}$ of them. The affair is this: that of the 7 metre-angles of convergence amplitude needed during work he must use only $\frac{1}{3}$ (or really somewhat less) or $2\frac{1}{3}$ metre-angles. In other words he must converge at the most for an object-distance of 43 *cm*, unless asthenopia shall arise.

But if his working distance for one or another reason must be precisely 33 *cm*, it will be our task to help him for this distance, without his being obliged to converge up to more than 43 *cm*, and this we can accomplish by means of prisms. The difference in the convergence between 43 *cm* and 33 *cm* expressed in metre-angles amounts to $\frac{2}{3}$ of a metre-angle

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[1] Bericht ueber die XVII. Vers. d. ophth. Gess. zu Heidelberg, 1885.

($\frac{1}{3}$ of the lacking 2 metre-angles) = 2.20°. Prisms 2^1° base inward for each eye would then enable him to work between the customary working distance and the convergence.

Practically the affair is not so easy as here described. Prisms are the most difficult things to order, because we can never predict what effect they will have. Many people cannot endure them, to others they are of the greatest assistance. If prisms seem indicated, I always tell the patient that they are chiefly experimental, and may be of no use to him.

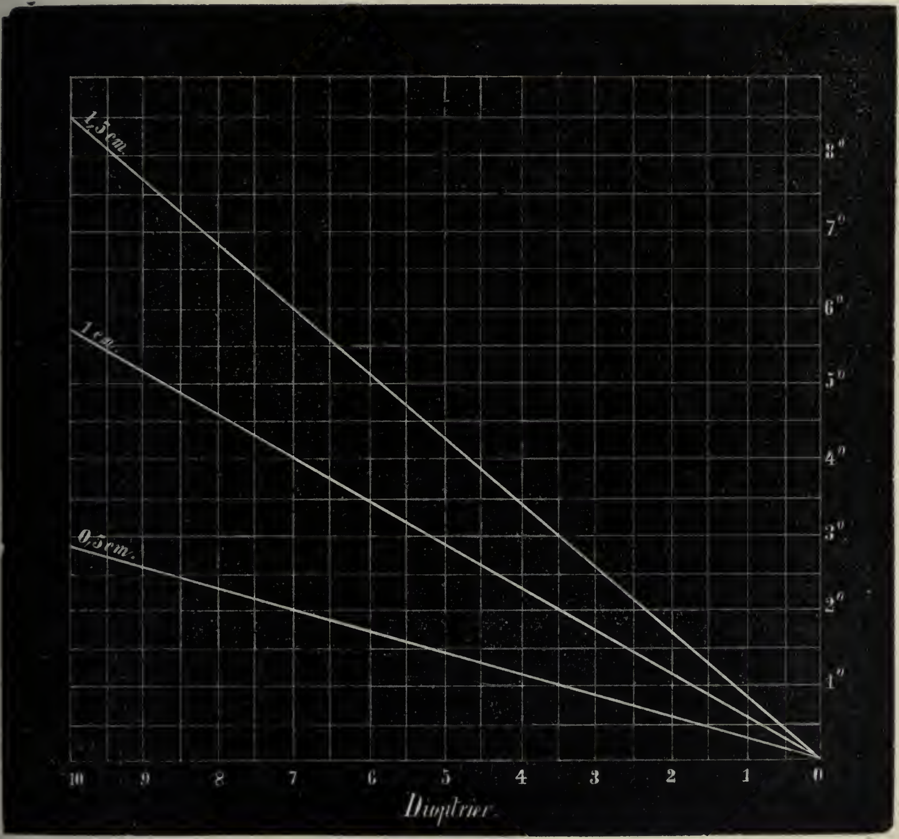

FIG. 7.

We should never begin with prisms stronger than 2° or 2½° for each eye; if necessary strengthen them later. Stronger than 4° for each eye can rarely be endured.

When we are obliged to use spherical lenses for presbyopia or refractive disabilities, we know that the use of concave lenses will oppose convergence insufficiency, so that it may often suffice to give the patient more or less correcting concave lenses for his work.

When convex lenses have to be worn (thus sparing the accommodation), the convergence is robbed of a good support. It may nevertheless occur, that when we remove the accommodative asthenopia by means of the convex lenses, the convergence asthenopia will be increased. Hence we must often attempt a combination of spherical lenses with prisms. And yet with stronger spherical lenses we can accomplish this effect by making the distance between the centres of the lenses different from that between the pupils.

The effect of decentring lenses can be seen from Fig. 7, where the three lines represent the prismatic action of spherical lenses in degrees (not prismatic degrees) with a difference of 0.05, 1, and 1.50 *cm* between the centres of the lenses and the pupillary distance. With + or — 2 D we can obtain an effect of 1° 9′ when the distance between the centres of the lenses is 1 *cm* greater or less than the pupillary distance. The effect will correspond to a prism of a little more than 1° for each eye.

We can scarcely utilize a difference of more than 1½ *cm*, and then there is nothing more to do than to produce this difference by shortening or elongating the nose-piece of the spectacles. In case of a greater difference than 8 *cm* we can produce the surplus by grinding the lenses eccentrically into the frames.

Second Group.—Divergent position of equilibrium for distance, latent divergent strabismus.

Here we find a fusion far point, corresponding to prism of 10° to 20°, according to the degree of the divergence. The fusion near point is pushed away, and yet in *young* persons we occasionally find a normal near point despite marked divergence.

The equilibrium test shows the upper ruler (prism base down for left eye) pushed more or less to the right at all distances. If we assume that in a given case at the distance of 5.75 *m*, the movement amounts to 50 *cm* (5 °), it would then be expected that the same degree of divergence would also be observed for shorter distances, but I have never found any such condition of things. Generally we find an increasing divergence for shorter distances, *i. e.*, *the latent divergence is generally accompanied with convergence insufficiency.*

11*a*) Student, aged 27, 90 ° — 0.5 — 2.5 ; 80 ° — 1 — 2

	$\frac{20}{30}$	$\frac{20}{30}$
3.45 *m*	Div. 36 *cm*	= 6 °
1.15 "	" 16 "	= 8 °
0.286 "	" 6 "	= 12 °

Fusion far point, prism 12 ° ; near point, 7 *cm.*

11*b*) Mrs. N., aged 52, 〃 〃 + 3.5 ; 〃 〃 + 4.5

3.45 *m*	Div. 40 *cm* = < 7°	With + 3.5 ; + 4.5
1.15 "	" 16 " = 8°	
0.286 "	" 5 " = 10°	

Fusion far point, prism 18 ° ; fusion near point, 10 *cm* (barely ; the eye generally deviates before reaching this distance).

There is no range of equilibrium in latent strabismus divergens, but simply a point of equilibrium which is negative, and rather near the fusion far point, and, as in the first case, often coincides with it.

When the muscles are normal, we have a range of convergence extending from infinity to the fusion near point, and an amplitude of divergence from infinity to the fusion far point. Infinity is the limit between these two ranges, because the static position of equilibrium is parallelism between the visual lines. Where the static position of equilibrium is divergent, the point of equilibrium forms the boundary between the range of convergence and divergence. Hence the range of convergence in those belonging to this group will be mostly extended beyond infinity to the fusion far point, whilst the range of divergence will be extremely slight if any. Therefore, the range of convergence will form almost the whole of the range of fusion, and where we find a normal fusion near point the amplitude of convergence will be largely increased. This seems to occur chiefly in younger persons, and suggests that the interni in latent strabismus divergens are better developed than usual, and must be very powerful, since for years, nay, often for life, they perform the majority of the work demanded of them under these conditions of equilibrium.

We find, however, still more frequently that the interni—earlier or later, depending entirely upon the degree of the divergence, the strength of the muscles, and particularly the alterations in the refraction of the eyes—can no longer perform their work; defective convergence ensues at once. This then gradually leads to the deviation of an eye, first at the ordinary distance for work, later in looking off at a distance, at all events in the higher degrees of the affection; then we may find a periodic squint, either only for near or often enough for all sorts of distances, and in this case the natural termination can be a manifest divergent strabismus.

When defective convergence begins, asthenopia must ensue; but when things have gone so far that one eye constantly deviates, and that squint is actually present for the ordinary distance at which the work is held, the former weariness disappears, and the patient no longer complains of diplopia. Double images may at first be seen, but they soon disappear.

The result of all this is, that here we have a peculiar condition, which arises from the difficult conditions of convergence. The patient gradually learns of his own accord to overlook the image of the deviating eye which falls upon the out-turned half of the retina. Such an individual cannot see crossed double images under ordinary circumstances, and the fusion will then be more and more dissolved.

We have the right to conclude that this condition is acquired, and not congenital, owing to the facts that the patient may have passed through a stage in which he actually saw double; that he can once more learn how to perceive the crossed double images; and finally, that when the retinal image is brought by prisms upon the inner portion, it is instantaneously embraced, as is proved by the homonymous double images. In this person, therefore, we shall easily be able to fix the situation of the fusion far point, whilst the fusion near point can only approximately be discovered because we have to watch closely to see when the eye deviates; it will therefore be best to employ a larger object, as a lead-pencil, for this part of the test.

When the refraction and the vision are about the same in the two eyes, now one eye will deviate and now the other, and in this case it will be impossible for the patient to see crossed double images; but when the condition of things is such as invariably to cause one and the same eye to deviate, we shall generally be able to bring out the double images, when we can induce the patient to fix with this eye, and to let the better eye deviate. Having never had an opportunity in the better eye of learning how not to see the images upon the outer halves of the retina, he will at once grasp them, and diplopia will arise.

I have sometimes noticed in cases of extreme latent divergence that the patient could not see double during the test for equilibrium, particularly at greater distances, in spite of the fact that the variation in the height of the objects was produced by a prism with the base down before the left eye, whereby the image in this eye was made to lie in a portion of the retina which otherwise was not simultaneously affected with the macula lutea of the other eye. In such a case it may happen that we can establish nothing but the fusion far point.

There was one patient whose fusion far point corresponded to a prism of 30°, in whom, however, the second image appeared, after so strong prisms with the base inward had been brought in front of the other eye that the double image lay at the same level with the macula. With prism 25° he saw two rulers at 3.45 *m*. At 0.286 *m*, however, he caught the double images without any difficulty; here was a divergence of 11 *cm* = 22°. He had myopia, 5.5 D, and read with — 4.5 D.

Finally, we meet with cases in which during the equilibrium test only a slight divergence is observed or none at all, or there is an actual convergence, although the eye diverges noticeably underneath the covering hand.

All this goes to show that the equilibrium test sometimes gives no results at all, and in others an incorrect result, but still I do not believe that we are justified in rejecting the test entirely, as some have proposed. For in most of the cases it is perfectly reliable, and under all circumstances we verify the result by other investigations.

Here are examples of the rarer forms.

12) Miss D., æt. 40. ″ ″ — 7 ; 178° — 1.5 — 6.
$\frac{20}{40}$ < $\frac{20}{40}$

She complains of burning in the eyes, and weariness on reading; extreme deviation under the covering hand; fusion far point prism 24°; fusion near point cannot be determined, because she cannot see crossed double images.

During the equilibrium-test she can with difficulty see two rulers, but when she sees them for an instant as two, she exhibits not a little convergence.

13) Miss B., æt. 23. ″ ″ — 1 ; ″ ″ ″ ″
$\frac{20}{40}$ $\frac{20}{40}$

Similar complaints as the above patient; extreme deviation under the covering hand. Fusion far point 22°, fusion near point 7 *cm.*

Equilibrium test shows the rulers above one another, with slight convergence; when one eye is covered it deviates greatly, but flies back again when the covering hand is removed. While divergent, the double images could not be perceived.

Latent divergent strabismus is seen at all ages, even in children, but it is rare for children to be brought to the oculist for this defect alone. Generally, the patients are from fifteen to thirty years of age, complaining of asthenopia or imagining that one eye deviates.

Can we think that this condition depends upon the fact that the static muscular relations really alter with the age? I think that they are congenital and never change but slightly. We can explain the rarity of divergence in children partly by the fact that their refraction is generally hypermetropic, and partly because the interni at that age are still powerful and have not yet been overburdened by the persistent and extreme amount of convergence demanded at school at their age.

It often happens that several children in the same family suffer from latent divergence, and so too we not infrequently see that when one of the parents suffers from latent divergent strabismus one or more of the children suffer with the same affection. This proves that the condition may be hereditary; and this again suggests that it is congenital.

14) Mrs. B., æt. 28. " " — 5 ; " " — 5.

Accustomed to close the left eye whilst reading; otherwise no trouble at close work; extreme divergence under the covering hand; fusion far point, prism 28°.

The fusion near point cannot be determined because she cannot perceive any crossed double images; she can converge with a lead-pencil up to about 20 *cm*; she also fails to see double images with the equilibrium tests.

She has always regarded the closing of her left eye in reading as a bad habit, which she thinks she inherited from her father, who always closed his left eye in reading.

We know, however, that closing one eye for near work is something that a few with abnormal muscles have had the good luck to learn how to do, and because the father also had this "bad habit" we can conclude that he had latent divergence.

This mother came with her son of five years, because after looking long at any thing there seemed a momentary squint, and I soon discovered that under the covering hand the eye actually diverged a great deal. He was, however, too young to test his equilibrium.

Latent divergent strabismus is observed in all conditions of refraction, but we find it most often combined with myopia and emmetropia. Hypermetropia is a rarer refractive condition in adults, and an hypermetrope will not be troubled so soon with a moderate degree of divergence.

In the *treatment* of latent divergent strabismus we have three points to bear in mind. In the first place *any difference* in refraction must be compensated for, and by accurate correction of any astigmatism, we must endeavor to make both eyes see alike in order to increase as much as possible the need for fusion.

The fusion will generally be more or less "dissolved," and therefore we must in the second place endeavor to strengthen it. For this purpose we may most advantageously resort to stereoscopic practice with decreasing distance (Javal). In using this the need for fusion will not only be awakened and increased, but the interni will be strengthened, and we have a right to think that these exercises may act as a sort of gymnastic upon the muscles. It is a fact, that the asthenopia in a few cases can only be alleviated to any great extent by stereoscopic exercises.

Thirdly, we have to direct our treatment directly against the divergence; this we do indirectly by prescribing concavo-prismatic lenses, or directly by tenotomy of the rectus externus.

If myopia is present there may be no need of any thing else than correcting lenses. The accommodation will then begin to play its part, and the support which the interni then obtain will, in many cases of moderate divergence, excite and maintain the necessary convergence. If concave lenses do not alone suffice, we can increase their effect by a combination with prisms. With Em, Pr and Hm, we can only oppose the divergence by prisms, which are to be used alone or combined with convex lenses.

15) Mr. P., æt. 38. 〃 〃 〃 ; 〃 〃 〃
$> \frac{20}{30}$ $> \frac{20}{30}$

He complains of burning in the eyes, and is constantly compelled to nip his eyelids in order to see clearly; the eyes deviate largely under the covering hand.

3.45 *m*	Div.	16	*cm*	=	< 3°
1.15 "	"	4.5	"	=	2.25°
0.286 "	"	3.75	"	=	7.50°

Fusion far point, prism 10°; fusion near point, scarcely 15 *cm*.

He was ordered for work Pr. 3° + 1; Pr. 3° + 1 base in; for out-of-doors Pr. 2°; Pr. 2° base in.

I rarely order prisms for a distance with so slight a degree of divergence as here, but, they were so agreeable, that he liked to use them; they made "objects look so firm and steady." He could work for a long time with the combination ordered for close work.

We must tenotomize the external rectus in the higher degrees of divergence, but we ought to know how great the effect of this tenotomy will be before we can decide whether it is the best thing to do in the given case.

V. Graefe gave very accurate rules for tenotomy in insufficiency, but I think that they are too complicated, and therefore I will report a few cases in order to show the amount of effect that I have discovered in my investigations.

16) Miss M., æt. 18 ″ ″ ; ″ ″ ″
$\frac{20}{30}$ $\frac{20}{30}$

3.45 *m*	Div. 80 *cm*	= 13°
1.15 "	" 32 "	= 16°
0.28 "	" 7 "	= 14°

Fusion far point, prism 26° ; near point, 7 *cm.*

Oct. 31 : Tenotomy R ext. rectus. Nov. 1 : 3.45 *m* — 0. This condition remained the same, *i.e.*, parallel equilibrium position for far.

Effect of tenotomy = 13° (Pr. 26°).

17) A. O., æt. 12. ″ ″ + 2 ; ″ ″ ″
$\frac{20}{50}$ $\frac{20}{30}$

Left eye deviates when reading ; no asthenopia.

3.45 *m*	Div. 120 *cm*	= 20°
1.15 "	" 44 "	= 22°
0.286 "	" 4 "	= 8°

Fusion far point Pr. 40° ; near point, 28 — 30.

Tenotomy L ext. rectus ; directly after at 3.45 *m ;* Div. 24 *cm* = 4°

Next day :	3.45 *m*	Div. 48 *cm*	= 8°
	1.15 "	" 16 "	= 8°
	0.286 "	" 1 "	= 2°

Fusion far point, Pr. 16° ; near point 8 *cm ;* effect of the tenotomy = 12° (prism 24°).

This is the first case in which I found divergence less for 0.286 *m* than at a greater distance ; it is remarkable that this condition remained after the operation.

18) Hanna O., æt. 10. ″ ″ ″ ; ″ ″ ″
$\frac{20}{20}$ $\frac{20}{20}$

5.75 *m* . . . . Div. 84 *cm* = 8.4°
1.15 " . . . . " 19 " = 9.5°
0.286 " . . . . " 5.5 " = 11°

Fusion far point, Pr. 16°; near point, 5 *cm.*

Despite the normal fusion near point she had considerable trouble on account of the divergence.

Tenotomy of left external rectus :

Next day : 5.75 *m* . . Conv. 52 *cm* = 5.2°
1.15 " . . " 7 " = 3.5°
0.285 " . . " 0.75 " = 1.5°
Later : 5.75 " . . " 52 " = 5.2°
1.15 " . . " 10 " = 5°

The conjunctival incision had as usual been closed with a vertical suture : this was removed and a broad horizontal suture inserted, and introduced so deeply from without that the muscle was grasped—and then we found :

5.75 *m* . . Conv. 5 *cm* = 0.5°
Two days later : 5.75 " . . Div. 16 " = 1.6°
1.15 " . . " 4 " = 2°
0.286 " . . " 1 " = 2°
Two months later : 5.75 " . . " 4 " = 0.4°
1.15 " . . " 0
0.285 " . . " 0

Fusion far point, Pr. 8° ; near point, 4 *cm.*

No difficulty in reading : effect of the tenotomy 13.5° (Pr. 27°).

19) Miss N., æt. 20. $5^\circ - \underset{\frac{20}{70}}{2 - 11}$; $5^\circ - \underset{\frac{20}{40}}{2.5 - 6}$.

Reads with RE : left at first held correctly but it soon deviates.

With lenses 5° — 2 — 10 ; 5° — 2 — 4 : we found at

3.45 *m* . . . . Div. 30 *cm* = 5°
1.15 " . . . . . . " 24 " = 12°

With lenses 5° — 2 — 9 ; 5° — 2 3.5, for 0. 286 *m* : Div. 4.5 *cm* = 9°.

Fusion far point, Pr. 24° ; near point, 55 *cm.*

The test for 1.15 *m* shows that the divergence for far must be greater than is given—to judge from the fusion far point, about 12°.

February 26.—Tenotomy of left ext. rect., after which :

3.45 *m* . . . . Conv. 16 *cm* = 2.5°
1.15 " . . . . " 4 " = 2°
0.286 " . . . . Div. 1 " = 2°

She complains of constant homonymous diplopia.

3.45 *m* . . . . Conv. 32 = 75°
1.15 " . . . . " 12 = 6°

A deep horizontal suture removed the convergence.

February 28.—

3.45 *m* . . . . Conv. 16 *cm* = 2.5°
1.15 " . . . . " 0 "

Glasses for out-of-doors : 5° — 2 — 7 ; 5° — 2.5 — 3.5.

March 25.—With the lenses.

At 3.45 *m*, 1.15 *m*, and 0.286 *m* no divergence—effect of the tenotomy 17° (Prism 34°).

20) Johanna, æt., 36 " " " ; " " "
$\frac{20}{20}$ $\frac{20}{20}$

Always inclined to see double : asthenopia well marked of late by artificial illumination.

5.75 *m* . . . . Div. 76 *cm* = 7.6°
1.15 " . . . . " 14 " = 7°
0.286 " . . . . " 6 " = 12°

Fusion far point Pr. 15° ; near point, 25 *cm*.

November 19.—Tenotomy L ext. rect., immediately afterward at :

5.75 *m* . . . . Div. 6 *cm* = 0.6°
1.15 " . . . . " 3 " = 1.5°
0.286 " . . . . " 2 " = 4°

November 28.—

5.75 *m* . . . . Div. 16 *cm* = 1.6°
1.15 " . . . . " 6 " = 3°
0.286 " . . . . " 1.75 " = 3.5°

Fusion far point Pr. 7° : near point 8 *cm*, no asthenopia.

She reported again in a few months an account of asthenopia by gaslight.

5·75 *m* . . . . Div. 44 *cm* = 4.4°
1.15 " . . . . " 11 " = 5·5°
0.286 " . . . . " 3·5 " = 7°

Fusion far point, Pr. 9°: fusion near point, 13 *cm.* Lenses ordered Pr. 2°; Pr. 2°: base in: effect directly after tenotomy = 7° (Prism 14°): reduced later to 3° (Prism 6°).

The result in these five cases was therefore:
In No. 16 = 13° (Prism 26°)
17 = 12° (" 24°)
18 = 13.5° (" 27°)
19 = 17° (" 34°)
20 = 7° (" 14°): finally only 3° (Prism 6°).

The effect in 18) and 19) would probably have decreased, but as the convergence was greater than desirable, I thought it most advisable to reduce the effect by the horizontal suture. Nevertheless, we see that with a fusion far point corresponding to prism of 26° to 28°, we can calmly tenotomize the rectus externus without fearing too extensive a result. With a fusion far point less than 26° and down to 16° there is no need of convergence after the operation, but we must be ready to limit the effect by a deep horizontal suture—*i.e.*, we must perform a slight advancement operation, and particularly in children. The older the patient, the more fearlessly we can tenotomize in the lower degrees of divergence, even when the fusion far point is less than a prism of 16°.

If convergence follows the operation it may be difficult to decide, whether we shall act at once or wait, because the over-effect begins to decrease within a few weeks; still on the day after the operation I should not like to permit a convergence greater than a few degrees.

There are, however, a few cases in which we cannot do much if any thing for our patients. This is especially possible when the divergence is too small for a tenotomy to be permissible, but still too great to obtain any correction by prisms, owing to a high degree of convergence-insufficiency.

In such cases I advise the patient to close one eye, or to cover it, as a few patients had already done of their own accord ; but this has seldom had the desired effect.

First Group.—Convergent position of equilibrium for distance or latent convergent strabismus.

Here we find the fusion far point pushed in corresponding to a fusion of 6° down to 0°, and the fusion near point nearer, or even normal or pushed outward, since latent convergent strabismus may be combined with a convergent insufficiency.

The equilibrium tests generally show convergence for all distances, usually decreasing somewhat at 1.15 and 0.286 *m*, but even at this distance there may be a greater convergence than at 3.45 *m*. Near the fusion near point, however, we come to a spot where equilibrium reigns, and within this distance divergence comes on.

In latent convergence, too, there is no range of equilibrium, but only a point of equilibrium, and this lies more or less in the neighborhood of the fusion near point.

21) H. D., æt. 11 " " " + 1 ; " " + 1
5.75 *m* . . . . Conv. 32 *cm* = 3.2°
1.15 " . . . . " 6 " = 3°
0.286 " . . . . " 1 " = 2°

Equilibrium at 20 *cm* ; fusion far point, Pr. 5° ; fusion near point, 6 *cm*.

22) M., æt. 25 " " — 1 ; " " — 1.5
$\frac{20}{20}$ $\frac{20}{20}$
5.75 *m* . . . . Conv. 16 *cm* = 1.6°
1.15 " . . . . " 3.5 " = 1.75°
0.286 " . . . . " 0.5 " = 1°

Equilibrium 40 *cm* ; fusion far point, Pr. 3° ; fusion near point, 6 *cm*.

23) Miss S., æt. 19 " " — 2.5 ; " " — 2.5
$\frac{20}{50}$ $\frac{20}{50}$
3.45 *m* . . . Conv. 56 *cm* = 79° with — 1.5 ; — 1.5
1.15 " . . . " 20 " = 10° without lens
0.286 " . . . " 3 to 6 " = 6° to 12° without lens

Equilibrium at 12 *cm ;* fusion far point = ∞ ; fusion near point = 10 *cm*.

Since the point of equilibrium lies close to the fusion near point, and forms the boundary between the range of con- and di-vergence, the range of convergence in this condition must be extensively reduced and that of divergence enlarged. The externi will have to do a greater share of the work, and therefore they must be very strong in order to maintain the standard demanded of them.

Just as we discovered in the previous group that convergence became defective when the interni weakened, so here, when the externi give way, defective divergence must ensue, and the fusion far point must be pushed inward. When it already lies inside of infinity, or falls within infinity on account of the defective externi, we have a manifest convergent strabismus for far, which must increase the more the externi lose in energy. We often hear of squint when the fusion far point lies precisely at the limit of infinity, or when squint has already been present, that it increases whenever the patient does not feel well or has a headache, due of course to the fact that one cannot work when unwell as when feeling well, and this is as true of the ocular muscles as of those of the rest of the body.

We ought in cases of manifest convergence for far, to expect homonymous double images, and as a matter of fact we do hear of them, but we see that here too the same state of things is developed as we have mentioned in divergence. The patient learns to overlook the image of the deviating eye, and as the eye glides inward, the images of the inturning retina are suppressed. For this reason in manifest convergence for far or where this condition is developing, it is often difficult or impossible to fix the fusion far point.

If the images pass over the outer halves of the retina they will at once be grasped; crossed double images appear, and the fusion near point can always be determined.

For these reasons we find a complete analogy between the conditions for the fusion near point in divergence and the fusion far point in convergence.

24) Miss J., æt. 19 " " " ; " " "
$< \frac{20}{20}$ $< \frac{20}{20}$

Scarcely visible manifest convergent strabismus for far.

3.45 *m* . . . . Conv. 50 *cm* = > 8°
1.15 " . . . . " 13 " = 6.5°
0.286 " . . . . " 2.5 " = 5°

Equilibrium at 16 *cm*:

The fusion far point cannot be determined because the patient cannot grasp the homonymous images. Fusion near point, 12 *cm*.

25) Mrs. K., æt. 51. 0° — 0.5° — 5 ; 0° — 1 — 5
$< \frac{20}{20}$ $< \frac{20}{20}$

The right eye turns a great deal in : patient thinks it did not do so till a few years ago, and that it has lately grown much more marked.

3.45 *m*. Separation of double images too great to be measured at this distance.

1.15 *m* . . . . Conv. 64 *cm* = 32°
0.286 " . . . . " 8 " = 16°

Unfortunately the equilibrium point was not then fixed : it probably lay at 8 to 10 *cm*. The fusion far point cannot be determined as patient cannot see homonymous double images. Fusion near point was not measured : *binocular reading at* 20 *cm!*

The convergence that appeared and even increased at such an advanced age can only be explained by taking it for granted, that the fusion far point had been pushed inward owing to the lack of tone in the externi which were forced to undertake a task beyond their power.

Where the vision of the eyes varies and the same eye always deviates, we can occasionally cause the patient (even with manifest convergence for far) to see double images, particularly when we can induce him to fix with the eye that generally deviates.

26) Ottilie, æt. 14. Periodic squint many years. Thinks as a child she could read without squinting ; now the right eye deviates inward whilst reading. For distance she arranges her eyes in two ways : one when she wants to see clearly, in convergence ; but when she wants to be "beautiful" she is satisfied with less acute vision and her eyes are straight. In the latter case,

S = $\frac{20}{70}$, but when she squints and looks with one eye alone (the left) S < $\frac{20}{20}$.

" " " + 1 ; " " + 1
$\frac{20}{20}$ $\frac{20}{20}$

With correcting lenses she sees equally well, whether she squints or not.

She always looks with the left eye, and never had double images ; but when she fixes with the right eye, which she easily learned, and the left eye deviates, homonymous images at once appear, because the image falls on the inner half of the retina of the left eye, and these images, from lack of a chance to overlook, she has not yet been able to get rid of.

3.45 *m*—when she squints—conv. 80–90 *cm* = 13°
1.15 " . . . . " 12–32 " = 6°–16°
0.286 " . . . . " 9 " = 18°

Equilibrium point at about 8 *cm* : fusion far point (fixation with right eye), Pr. 8°, base in. Fusion near point, 8 *cm*. With + 4 ; + 4 she reads without squinting, and for 0.286 *m* has converg. 1 to 2 *cm* = 2° to 4°.

The two following cases are interesting on account of the high degree of myopia :

27) Dr. K.. age 55. " " − 14 ; " " − 11
$\frac{20}{50}$ $\frac{20}{40}$

3.45 *m* . . . . Conv. 64 *cm* = 10½°
1.15 " . . . . " 8 to 20 " = 4° to 10°
0.286 " . . . . " 0.5 " = 1°

Equilibrium point at 20 *cm ;* fusion far point ∞ ; fusion near point, 7 *cm*. In the equilibrium test he can only with difficulty maintain the double images ; that of the left eye disappears easily ; he asserts that he could voluntarily suppress it.

28) Frederick K., æt. 19. " " − 14 ; " " − 14
$\frac{20}{100}$ $\frac{20}{100}$

No squint, but under the hand the eyes deviate excessively inward.

1.15 *m* convergence about 40 *cm* = 20°.

He reads binocular at about 12 *cm*, and at this distance there is apparent equilibrium.

Latent convergent squint occurs in all conditions of refraction. Most of the cases are, however, myopic, and the myopia probably explains why most of the cases are latent. With Em or Hm a *manifest* strabismus would in all probability long since have developed.

The significance of Hm can be seen from this case :

29) Mr. H., æt. 27. ″ ″ + 4 ; ″ ″ + 3
$<\frac{20}{40}$ $<\frac{20}{30}$

	without lens :	with + 4 ; + 3
3.45 *m* . .	Conv. 32 *cm* = >5°	Conv. 8 *cm* = >1°
1.15 “ . .	“ 20 “ = 10°	“ 2 “ = 1°
0.286 “ . .	“ 4–5 “ = 8°–10°	Div. >0

Equilibrium not tested.	Probably about 30 *cm.*
Fusion far point, Pr. 16°, base *out.*	Pr. 3°, base *in.*
Fusion near point, 18 *cm.*	20 *cm.*

Without a lens, convergent strabismus with diplopia ; with lens, fusion.

30) Betty P., æt. 12. ″ ″ + 5.5 ; ″ ″ + 5
Ophthalmoscope, 5 to 6 D Hm.

	Without lens :	with + 4 ; + 4	with + 6 ; + 6
5.75 *m* . . .	Conv. 11°	Conv. 6°	Conv. 0.4°
1.15 “ . . .	“ 13°	“ 6°	“ 0.5°
0.286 “ . . .	“ 10°	“ 6°	“ 0

Equilibrium at 16 *cm* : not tested : 28 *cm.*
Fusion far point, Pr. 5°, base in.
Fusion near point, 6 *cm.* 75 *cm.* 75 *cm.*

A slight convergence is actually present in both of these cases, although the values with + 6 ; + 6 in the latter seem to be a little too small. In the use of such strong convex lenses we must accurately arrange the distance between the centres of the lenses according to the pupil distance, on account of the considerable prismatic effect. The patient has probably been wearing the proper lenses in a frame that was better adapted for a grown-up person.

The approach of the equilibrium point is the reason why, in hypermetropia with convergence, the externi have so much difficulty in performing their task.

In Case 30 we really have an equilibrium point at 28 *cm*, corresponding to 12°, or to a convergence of 3.5 metre angles, but on account of the Hm the externi are compelled to work with the equilibrium point at 16 *cm*—*i.e.*, with a convergence of about 21°, or 6 metre-angles.

When we see how difficult are the conditions in Hm, we need not be surprised that the latent convergence which occurs at all ages is most often seen in childhood, and then so easily changes into manifest convergent squint. Hypermetropia is the most frequent condition of refraction in childhood, and what is of some importance, surely, the fusion is not so well exercised in the early years of life as is the case in adults.

The treatment has already been suggested under the head of divergent strabismus. Convex lenses are used instead of concave, and if prisms are available they must be used with the base in instead of out as in divergence. On the whole, the essentials of treatment are the same in both.

Most of our patients in this class are children, but we do not see them until they have already developed a periodical squint and got rid of their diplopia. If the periodic squint is still in its developmental stage, we may be able, for some little time, at least, to obviate it by carefully covering the good eye with a shade, and only letting the child look with the eye that has habituated itself to turn inward. He will then be compelled once more to occupy himself with the images formed in this eye, and those halves of the retina whose images were suppressed will again be exercised. If the shade is removed after ten days or a fortnight, fusion will generally be observed, and if it is actually obtained the periodic squint must not have lasted for any length of time.

It may be useful to continue this method of treatment for some time, and then, at a later date, to tenotomize the internal rectus.

If we are actually able, in such small patients, to measure approximatively the degree of the convergence, *i.e.*, the location of the point of equilibrium, or can convince ourselves that the convergence is great enough to authorize us to perform a tenotomy, it must be the most rational step to operate in this stage, and we shall be more certain of obtaining the desired fusion.

Having thus inquired into the conditions in latent convergent strabismus, I believe, from what has already been said, and from the examples that have been cited, that latent and manifest strabismus are one and the same affection. When the manifest squint develops itself from the latent, the range of fusion will become more or less rapidly limited, so that finally there remains nothing but a point of fusion coinciding with the point of equilibrium.

The degree of the deviation is, of course, the deciding influence whether it shall remain latent or become manifest. If the divergence or convergence is well marked, manifest squint will develop under all circumstances, even if it were not already present from the birth of the child.

In the slighter degrees of deviation other influences come into play, *e.g.*, the refraction, and, more than any thing else, any condition of things that causes a marked variation in the sight of the two eyes, such as anisometropia, congenital amblyopia, maculæ of the cornea, etc., etc. When, among innumerable children who have had phlyctenula of the cornea, we see a few in whom a latent strabismus is developed, it is hard to imagine any thing else than that this must have been present a long time before, but which can now no longer remain latent simply because the image in one eye has become too ill-defined for fusion to be maintained. I once saw a case in which, during an affection of the periphery of the cornea, with a slight shadow over the pupillary field, there ensued a convergent squint, which disappeared again when the affection retrograded and the cornea became clear.

I think that statistics will show that convergence and divergence occur with about equal frequency. The question then will be: Why is convergence so frequent in childhood and divergence so late in its appearance, and why does convergence in children so easily transmute itself into manifest squint, whilst the divergent squint, at least according to my experience, in adults more easily remains latent?

These questions have really been answered. Children are generally hypermetropic, and when convergence is present beforehand the point of equilibrium will move so far inward that in many cases the externi cannot maintain fusion for any length of time, and the strabismus occurs. This will happen all the more easily in children, since their power of fusion has not been so thoroughly worked up, nor is it so strong, as in adults.

The condition of things is different in latent divergence. Here the hypermetropic refraction in childish years forms an equilibrium against the abnormal muscular relations, and only with increasing age, when the refraction alters, the Hm passes over into Em or My, and when the interni, overtaxed, grow weary from continuous exertion, then comes to light the divergence as a latent, or rather, perhaps, as a manifest, strabismus.

EMBOLISM OF THE SUPERIOR NASAL BRANCH OF THE ARTERIA CENTRALIS RETINÆ.

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(*With two wood-cuts.*)

WHILE acting as clinical assistant under Mr. Gunn in the Royal Ophthalmic Hospital last year I had an opportunity of examining almost daily for three weeks the following case. The only interest that attaches to it lies in the fact that most published cases and most pictures of partial retinal embolism refer one to those changes in the fundus which occur long after the accident has taken place, while in this instance I was able to examine the patient early, and to continue the examination daily thereafter for the period mentioned.

Forty-eight hours before I saw him, H. W., aged twenty-three, clerk, was talking to a friend in the street, when without any warning, he suddenly "lost all the sight in his left eye." He had neither before nor after any sensation of heat or fulness in his head; no vertigo, and no nausea. A few hours after the attack he began to see a little with the affected eye, and the next day could see very well. He applied for advice simply to ask whether he might, without danger, resume work.

He presented the appearance of a healthy young man, and did not complain of any thing except slight shortness of breath on exertion.

When seven years old had an attack of acute articular rheumatism, and since then four other seizures. Each time he was laid up in bed for several months, the last attack taking place nine years ago. On examining his heart found that he had a hypertrophied left ventricle, while a loud systolic bruit, propagated upwards, could be heard at the mid-sternum. There was well-marked increase of arterial tension.

R. E. V. = $\frac{6}{60}$; $\bar{c}$ − 2.50 D sph. = $\frac{6}{6}$ and Jaeger i.
L. E. V. = $\frac{5}{60}$; $\bar{c}$ − 3 . D sph. = $\frac{6}{6}$ and Jaeger i.
Both eyes = T. n and p. r. n.

Ophthalmoscopic examination of R. E. shows nothing abnormal except a small myopic crescent. L. E., fine floating opacities in vitreous, which make it difficult to see some of the fundus details

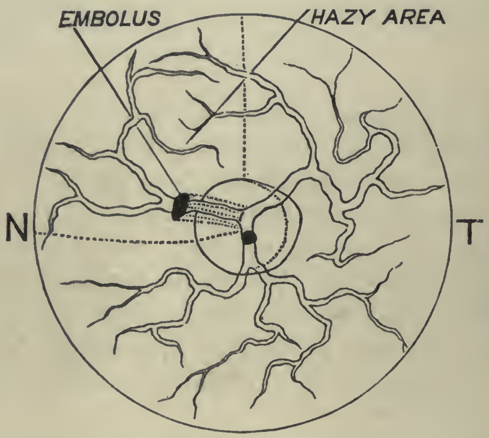

FIG. I.—LEFT EYE.

with distinctness. The upper and inner quadrant of the retina is hazy, but there are no gross changes in this locality. The macula has the normal appearance. The inner half of the O D is redder than the outer, and on its temporal border is a small crescent. There are no hemorrhages anywhere to be seen. Half a disc-diameter from the edge of the papilla, in a direction up and in, is a small, well defined yellowish spot. It lies immediately over, or at any rate obscures, the outline of the *ramus nasalis superior* at that point. The central artery divides about the centre of the disc into two branches, and the division of one of the latter into the usual superior nasal and temporal twigs takes place before the edge of the O D is reached. The whitish spot referred to is accordingly situated in or about the arterial twig one disc-diameter from its junction with the larger branch, as is roughly shown (arteries only) in the accompanying diagram. That portion of the vessel which lies between the yellowish-white spot and its origin on the papillary surface has a reddish outline, and seems to be swollen. In this situation it has lost its peculiar arterial reflex, and looks as if it might contain a blood-clot. On admission the field of vision for white objects was carefully taken, and a sector-like defect, corresponding generally to the hazy retinal quadrant, was mapped out. A McHardy perimeter was used with the 5 *mm*

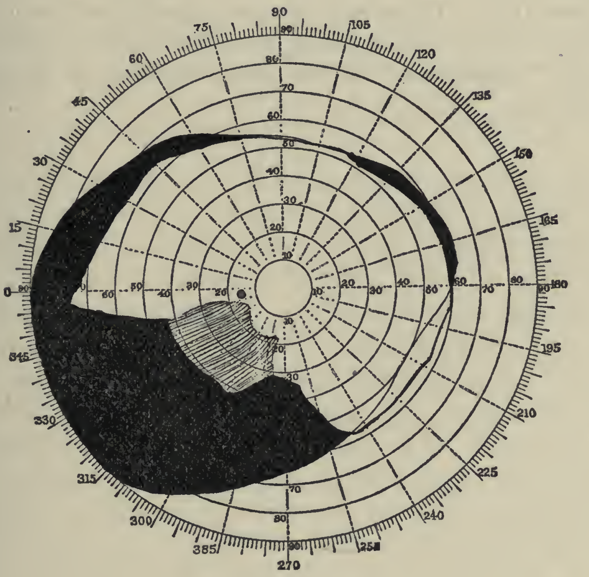

FIG. 2.

object. It will be noticed that an irregular area of indistinct vision for white (no color perception) extends from about the fortieth to the fifteenth parallel in the lower and outer quadrant of the chart. Outside of that, most of the quadrant shows absolute loss of perception of white. The field was taken several times at intervals, and no essential alteration from the result given was noted while the patient was under observation.

A week after the first examination the contour of the vessel between the whitish spot and the papilla is better defined, and the artery itself appears lighter in color, and smaller than before.

Neither the arterial trunk nor its other branches are affected as to color and size. It is doubtful whether the corresponding *vena nasalis superior* is swollen. The œdema continues about the same.

When last seen a clearer picture of the background could be had ; the limits of the retinal haze were quite as pronounced, and they had not extended. The coloring of the choroidal epithelium over the affected area was not visible ; a grayish appearance had taken its place.

Patient has apparently not discovered the absolute scotoma affecting his lower-outer visual field ; or, at least, he has made no complaint of it. There have been no appreciable changes in or about the macular region during the three weeks of observation, nor any "streakiness" of the retina outside of the area of grayish haze.

A REPORT OF FOUR OPERATIONS FOR CYSTICERCUS INTRAOCULARIS.[1]

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Translated by Dr. Ward A. Holden, New York.

THE clinical picture of intraocular cysticercus was left us by Albrecht von Graefe as almost complete, while the chapter on the treatment of this interesting affection—we speak of the operative treatment only, the medicamentous having proved worthless—remained still unfinished at the time of his death. Von Graefe obtained very satisfactory results in the extraction of cysticerci from the vitreous, those, however, in the deepest parts of the eye, especially the subretinal, seemed to him unsuited for operation.[2] In this direction we have advanced considerably in recent years. To Alfred Graefe is due the credit of having first grasped the idea and indicated the method of removing cysticerci from the deepest parts of the eye, making possible the safe extraction of this so pernicious guest, even from the posterior pole. Besides those described by von Graefe[3] there are on record a few cases of extraction of a cysticercus from the region of the macula lutea,[1] and the report of a successful operation of this sort seems permissible. I shall take this opportunity to present also three other observations of intraocular cysticerci, all of which were operated on.

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[1] From vol. xv., German edition, 1885.

[2] Von Graefe, in his last publication on intraocular cysticerci, in 1866 (*Graefe's Arch.*, vol. xii., p. 190), declared unsuited for operation those cases in which the entozoön lay behind the retina, near the posterior pole. It is interesting to know that he intended, at his first observation of cases of this sort, in 1854 (*Graefe's Arch.*, vol. i., p. 462), to go into the vitreous with a cataract needle, under the guidance of the ophthalmoscope, and kill the entozoön. As far as I have seen, he never carried out this idea. Much later such an operation was performed by von Wecker (*Handbuch von Graefe-Saemisch*, vol. iv., p. 711); it was unsuccessful, and the cysticercus was removed through a scleral incision.

[3] *Graefe's Arch.*, vol. xxiv., 1, pp. 209–232; vol. xxiv., 3, pp. 267–273; vol. xxviii., 1, pp. 187–202.

The *first case* was one of *cysticercus in the anterior chamber*, which I diagnosed in the right eye of Mrs. Wilhelmine Buntrock, aged thirty-four, on September 14, 1881. The patient had first noticed the change in the eye eight weeks before. There were no cysticerci on the surface of the body, and she was free from tapeworm; her husband, however, had a tapeworm. The patient counted fingers at eighteen feet; the disturbance of vision was due as well to a brown deposit on the anterior lens capsule as to the presence of the cysticercus, which was the size of a small pea. Changes of form and of position were observed, the head for the most part being drawn in. On September 17th, under narcosis, I extracted the entozoön, which lay at the bottom of the anterior chamber. Close to the sclera I made a linear incision 6 *mm* long with a Graefe knife, holding this as in dividing a corneal ulcer; in this way it was possible to avoid wounding the cysticercus which rested on the posterior surface of the cornea. It came out intact with the aqueous humor; a small portion of the iris prolapsed and was excised. The microscopic examination showed the ordinary characteristics of the cysticercus cellulosæ. The patient was dismissed cured seven days after the operation. The deposit on the anterior capsule of the lens disappeared gradually under the use of iodide of potassium. *On March* 29, 1882, *the vision of the operated eye was normal.* I saw the patient again some months ago, and at that time the eye was sound excepting the narrow downward coloboma of the iris.

The *second* patient, Mrs. Baitsch, aged forty-two, was received on December 13, 1881, with a *cysticercus in the vitreous of the left eye adherent to the bulbus wall* in the æquatorial region. The patient had noticed dark spots in the visual field of the left eye since June, and since November a progressive failure of vision. In the *visio directa* was a nearly absolute scotoma of 6° radius, the visual field was wanting inward, contracted above and below,

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[1] By Becker, at the Heidelberg Ophthalmological Congress of August, 1878 (quoted after Hirschberg, Eulenburg's *Realencyclopädie*, vol. iii., p. 609), then by Cohn (*Centralbl. f. Augenheilk.*, 1878, p. 145), and by Manz (*Arch. f. Augenheilk.*, vol. xiii., pp. 198–202).

and normal outward; the color limits were normal outward; fingers were counted eccentrically at four feet. The anterior segment of the eye was of normal appearance; at the posterior pole of the lens was a small translucent opacity; in the vitreous were a number of waving membranes, partly glistening white. On ophthalmoscopic examination the fundus appeared veiled, its lower half and the disc normal, the outer half of the retina detached. About six disc diameters from the nerve entrance was an intensely white irregularly outlined patch two and a half discs large; the lateral edges of this patch were prominent, most markedly the temporal, which projected into the vitreous as a sharp ridge, and spread out fan-shaped above into the retina. The patch was covered with irregular vessels, none of which passed beyond the sharp ridge; near by was a hemorrhage. When the eye was turned outward as far as possible and at the same time some 30° upward, the observer saw a cysticercus vesicle four discs in diameter, with a sharp convex contour, projecting far into the vitreous. On its surface were a number of small gleaming spots, and beside these a larger one unusually white, probably the head. No movement of this latter could be seen, although there was a wavy motion of the surface and of the edges of the entozoön; no vessels were present. The cysticercus, which was also visible by oblique illumination, was found six days constantly in the same position, and must therefore be considered to have been fixed at this point.

As regards the development of this affection, I believe that the white spot upward and outward from the disc represented the former sub-retinal position of the parasite, and that it had passed through the tent-shaped vascular position of the retina, and so reached the vitreous.

The surface of the patient's body was free from cysticerci, she had never noticed symptoms of tapeworm; a daughter, however, had suffered from tapeworm five years previously. The patient was in the seventh month of pregnancy, yet fearing further changes from a delay, the operation was made on December 19, 1881, under narcosis, and no consequences prejudicial to the general condition followed. Having rolled the bulbus downward and inward, I made a meridional incision with an ordinary bistoury between the rectus superior and the rectus externus, nearer the latter, first cutting through the conjunctiva, then the capsule of Tenon; the incision began 4 *mm* from the corresponding corneal margin, and was 7–8 *mm* long. The sclera lay bare; I then passed a Graefe knife with its back to the sclera (as in splitting the cornea) into the æquatorial end of the wound, made the counter puncture at the corneal end, and completed the section with a sawing motion; *the systicercus followed the knife directly*, it lay between the lips of the wound and was easily removed with iris forceps. No vitreous or fluid of any sort escaped from the incision. The slightly gaping wound in the capsule of Tenon and conjunctiva was closed with a suture. The wound was healed by the 27th; the ophthalmoscope showed the wound cicatrix as a narrow white stripe. A year later I found to my surprise that the posterior polar cataract and the opacities of the vitreous had entirely disappeared; central vision had not been regained, fingers were counted eccentrically at five feet.

The *third* case was of *subretinal cysticercus at the posterior pole of the eye.* Mrs. Johns, aged fifty-eight, consulted me January 22, 1884. She had noticed a visual disturbance in the left eye fourteen days before, and eight days before had found the eye nearly blind. The external appearance of the eye was normal, the ophthalmoscope showed translucent punctate and thread-like opacities deep in the vitreous, and in the macular region beneath the slightly changed retina an easily recognizable cysticercus; it was not regularly round, its horizontal diameter measuring five discs, its vertical somewhat less. The nasal margin of the entozoön lay one and a half discs distant from the nerve entrance, the lower margin close to the arteria temporalis inferior, the upper margin one disc distant from the arteria temporalis superior. The retina was detached for a short distance outward from the cysticercus, but appeared to lie close on the surface of the entozoön. From this it must be inferred that the entozoön had previously lain farther to the temporal side and had pushed itself toward the disc. The remainder of the fundus showed no changes. The fields for white, blue, red, and green had normal limits, there was a large central scotoma corresponding to the form of the cysticercus, extending upward 30°, downward 10°, inward 30°, and outward to the blind spot; movement of the hand was recognized within the scotoma. On January 25th the patient was operated on, no change having occurred in the cysticercus. It was proposed in the operation to lay the sclera bare at least to a point 21 *mm* in a direct line from the temporal margin of the cornea; at this point might be supposed to lie the temporal edge of the cysticercus, following the rules laid down by Alfred Graefe, considering the sagittal diameter of the bulbus 24 *mm*, and the half basis of the cornea 5.8 *mm*. In this case I concluded that the given distance was rather too large than too small, the eye axis probably measuring, on account of its hyperopia, less than 24 *mm*. On this ground it might be supposed that the extraction of cysticerci from the deepest parts of the eye would be more easily performed in hyperopic eyes than in myopic.

I began the operation, under narcosis, by dividing the rectus externus, loosening as little as possible its covering conjunctiva, and leaving a bit of tendon attached to the sclera as a guide for the location of the horizontal meridian; the rectus externus was armed with a thread, and then with blunt Cooper's scissors the connection between tunica vaginalis and sclera was loosened in the horizontal meridian as far back as possible, while the bulbus was rotated toward the nose. When the distance between the temporal margin of the cornea and the deepest free-lying point of the sclera was 15 *mm*, measured by the compasses, I made with straight scissors a cut 2 *mm* long through the conjunctiva and capsule of Tenon, which were dissected up, and let my assistants catch the margins of the wound with two small hooks and draw them to the temporal side, while at the same time the bulbus was fixed near the *temporal* margin of the cornea and rolled toward the nose. The sclera lay bare for 19 *mm*; I then passed a Graefe knife, its back to the bulbus, through the sclera far back in its free-lying portion, directed the knife toward the posterior pole, and made a cut 5 *mm* long. The wound did not gape, I had not carried the knife deep enough, and the sclera, as it appeared, was only superficially divided. The cut was so far back that after a slight displacement of the bulbus it could not be found again; I made then a second cut slightly beneath and parallel to the horizontal meridian, passing in the knife 1½ *mm* from the posterior end of the free-lying portion of sclera so deep that I was sure that this had been perforated, made a counterpuncture 4 *mm* farther back, and completed the section with a sawing motion; the cysticercus not presenting itself, I did not go in at once with the forceps, but widened the wound posteriorly with the straight scissors, since it appeared too small; *when the scissors were withdrawn the cysticercus lay in the wound. Neither vitreous nor fluid escaped from the wound during the operation.* The extracted body, as in the preceding case, was verified as a cysticercus by microscopic examination.

The tendon of the rectus externus was attached near the cornea, and the wound of the conjunctiva closely sutured. The wound healed well, and the patient was discharged February 3d, nine days after the operation. *With the ophthalmoscope no cicatrix could be seen.* On March 22, 1885, the patient counted fingers at two feet. With the ophthalmoscope were seen a number of small spots of pigment near the nasal margin of the disc, arranged parallel to it, and at the former seat of the cysticercus a large white patch with pigmented margins.

The *fourth* case was one of *free-moving cysticercus in the vitreous.* Mrs. Quedenfeld, aged forty-nine, presented herself June 10, 1884, stating that for three weeks objects seen with her right eye had appeared clouded ; the left eye was slightly hyperopic and astigmatic, otherwise normal ; in the right eye the vitreous was filled with five opacities, the fundus was correspondingly clouded but apparently normal, excepting a white patch four discs large upward and a little outward, near the boundary of the ophthalmoscopic field ; the view was too indistinct to determine whether vessels were present ; from the temporal margin of the white patch ran directly downward a bright narrow stripe, apparently elevated. V = $\frac{1}{3}$ after correction of hyperopic 3 D ; the visual field corresponding to this patch is contracted in the 180th meridian to 50° and in the 200th meridian to 35° (Förster's new diagram), elsewhere the fields for white and for colors are normal. I soon conceived the idea that this white patch was not a simple choroideal exudate, and the white stripe recalled the former case of cysticercus ; no movement could be seen however. On August 14th, the patient returned saying that for several days she had noticed a dark blotch before the eye ; at first glance one saw a large naked green cysticercus with iridescent borders ; the long slender neck with the head was stretched downward, and, like the vesicle itself, was in active motion.

The cysticercus had apparently developed between the choroidea and retina, then pushed itself toward the ora serrata. For this reason the ophthalmoscope did not at first discover it ; the white patch represented its former position, the white stripe its course between choroidea and retina. Finally it had left its concealment under the retina, had perforated this, and had passed into the vitreous.

The operation was performed at once. The patient was chloroformed in a half-sitting posture. After the introduction of the speculum I seized the bulbus in the vertical meridian and rotated it upward. The distance between the margin of the cornea and the limit of visible sclera was 16 *mm*, as measured by its compasses; I divided the conjunctiva and capsule of Tenon 3 *mm* outside the vertical meridian and parallel to it, until the sclera lay exposed. In cutting through the bulbus wall I carried the knife in the manner stated, pushing it, however, from above downward, as I should not have been able to sink it sufficiently if I had inserted it near the æquator. The wound was about 7 *mm* long, 6 *mm* from the margin of the cornea, and gaped sufficiently; normal vitreous stood in the wound but did not prolapse. I passed the forceps into the vitreous in various directions but did not succeed in seizing the cysticercus, which just before the operation had lain in the position stated above; I concluded that it had passed the boundaries of the ophthalmoscopic field. A second attempt to grasp the cysticercus failed; I then gave up the operation, which had reached the allowed limit, put one catgut suture in the sclera and two in the conjunctiva, and bandaged both eyes. On the 17th an ophthalmoscopic examination *revealed the cysticercus lying upward and outward.*

On October 1st the eye appeared normal externally, the vitreous, however, was much more clouded, and the cysticercus could not be seen; the patient distinguished only the hand, and perception of light was abolished in the upper field. In the middle of October the right eye appeared a little smaller than the other, it had become suddenly soft; the patient complained of pain in the whole right side of the head, and later of flashes of light in the left eye and inability to use it. In the beginning of November a yellow spot appeared low down in the vitreous, visible even by daylight, and as vision was almost entirely lost and ophthalmia sympathetica was feared, the eye was enucleated, November 2d; the left eye remained sound.

It struck me at once on looking at the enucleated bulbus that the cicatrix was drawn in at one point, while in life it had shown no such change. It seemed desirable to have the bulbus examined for micrococci; the investigations of Deutschmann have made it seem probable that the so-called ophthalmia sympathica arises from the migration of cocci from the irritating eye; I suspected that the enucleated eye might have caused ophthalmia sympathica. Professor Baumgarten had the kindness to carry out this investigation, and to present me with the following report:

"Externally the enucleated bulbus shows on its under surface a shallow radiate cicatricial retraction of the sclera of about pinhead size, several millimetres from the corneal margin. The bulbus is soft. After dividing it by an æquatorial section with a sharp razor, a funnel-shaped total detachment of the retina is seen; the space between choroid and retina is occupied by a clowdy-brown fluid. Corresponding to the scleral cicatrix the detached retina is adherent to the two outer coats of the bulbus. The detached retina encloses a soft yellowish-gray mass, the altered vitreous, the inner portion of which forms the capsule for a large, well-preserved cysticercus vesicle 11 *mm* in diameter. The inner surface of this vitreous cavity harboring the cysticercus has macroscopically the appearance of a muco-purulent substratum, but on microscopic examination it proves not to be a purely free purulent exudate, but a purulently infiltrated partly fatty degenerated granulation tissue. Dry cover-glass preparations of this tissue show under a homogeneous immersion objective (Zeiss $\frac{1}{12}$, oc. IV.) a few small micrococci, mostly single, some in pairs.

"Microscopic sections through the scleral cicatrix and the adherent neighboring inner part of the eye show in the former the presence of a typical cicatricial tissue, whose fibres cross the connective tissue fascicles of the sclera at right angles. This cicatricial tissue joins internally a new-formed fairly vascular connective tissue filling a defect in the choroid equally large with the scleral cicatrix, then passes through the retina, replacing its specific texture more or less completely, and continues into a richly vascular granulation tissue of transformed vitreous. In this latter lie embedded a number of well-formed hæmatoidin crystals and granular hæmatogenous pigment; the inner layers of the metamorphosed vitreous bounding the cysticercus are crowded with close-lying fatty-degenerated pus corpuscles. None of the usual methods show any sort of bacteria in section preparations of the cicatricial tissue of the sclera, choroid, and retina, nor in sections of the transformed vitreous. The same is the case with the opticus and its sheaths, which show nothing that might be considered a deposit of bacteria. Nor is this an abnormal cellular infiltration of the opticus and its sheaths."

The failure in extracting the cysticercus in the last case may be attributed to its movement in the vitreous. Had the entozoön kept the position which it held just before the operation I am convinced that the extraction must have succeeded.

My three cases of cysticercus in the posterior segment of the bulbus show, by a curious chance, the **three varieties** which Alfred Graefe distinguished as regards the **prognosis of the operation,**[1] viz., (1) a subretinal cysticercus, (2) a cysticercus adherent to the bulbus walls, and (3) a cysticercus in the vitreous. My small material confirms the experience of this investigation that the cysticercus of the last-named variety offers the least chances for a successful operation.[2] We may proceed more confidently to the extraction of a cysticercus in the region of the macula lutea than to the extraction of one freely movable in the vitreous. This sounds at first strange, yet it is supported not only by theoretic reasons, but also by the experiences of Alfred Graefe and those here reported.

In this respect the results which Albrecht von Graefe obtained in extraction of cysticerci from the vitreous are remarkable. In his last comprehensive publication on his experiences with intraocular cysticerci,[3] von Graefe states that since his last report (*Arch. f. Oph.*, vol. ix., 2, p. 180) he "has never made the there recommended æquatorial incision without bringing out the entozoön fully intact." He says,[4] two years later, in a publication "on the wider application of the peripheral linear incision": "Since I have employed the peripheral linear incision almost exclusively, in a period of nearly two years, I have brought out the entozoön fully intact in almost every case." In spite of this statement I cannot recommend, in place of the simple meridional incision, excision of the iris and extraction of the sound lens for the removal of a movable cysticercus in the vitreous. The linear incision carries with it, according to experience, the danger of causing sympathetic inflammation in the other eye,[1] a danger which demands special consideration, since eyes blind from cysticercus might still cause an ophthalmia sympathica.

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[1] *Graefe's Arch.*, vol. xxviii., 1, p. 189.

[2] Alfred Graefe had, out of twenty-four cysticercus operations eight unsuccessful results, and of these eight cases seven were cysticerci in the vitreous (*Graefe's Arch.*, vol. xxviii., 1, p. 189).

[3] *Graefe's Arch.*, vol. xii., 2, p. 190.

[4] *Graefe's Arch.*, vol. xiv., 3, p. 145.

Regarding the location of the meridional incision for the extraction of movable cysticercus in the vitreous, we may learn from von Graefe's operations, the results of which were more favorable than those of the method now in use, that the meridional incision should be made at such a place that traction instruments may be most readily introduced into the interior of the eye (below and outward, or below and inward).[2]

While the prospect of the successful extraction of a subretinal cysticercus, or of one adherent to the bulbus wall, is so much the greater the more exactly one strikes the seat of the parasite, there is no purpose in incising the bulbus at a point corresponding to the cysticercus when it is movable, as one cannot be sure that it will keep its former position after the incision is made. Secondly, it may be recommended to carry in the traction instruments after the bulbus is opened, under the guidance of the ophthalmoscope. If the operator gets the cysticercus in view—as may mostly be the case—he renders himself independent of the caprices of fortune to which otherwise he is subject. It seemed to me justifiable to call attention to these last two points, although I have no experience as to their practical utility; possibly with attention to them a favorable result might be obtained in cases similar to mine.

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[1] The possibility of ophthalmia sympathica after a meridional incision must be granted; a consequence of this sort has not, to my knowledge, been observed however. If ophthalmia sympathica be an infectious disease, as Deutschmann has made it seem probable, we can in no better way guard against this unfortunate accident than by treating antiseptically the wound so far as this is possible in the eye.

[2] *Cf.* von Graefe, *Graefe's Arch.*, vol. xii., 2, p. 188, and Hirschberg, Eulenburg's *Realencyclopädie*, vol. iii., pp. 605 and 606.

A CASE OF SUCCESSFUL EXTRACTION OF A CYSTICERCUS FROM THE VITREOUS WITH THE AID OF THE OPHTHALMOSCOPE.[1]

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Translated by Dr. Ward A. Holden.

AN unsuccessful attempt at extraction of a free-moving cysticercus in the vitreous, which I reported in 1885, showed me clearly the unreliability of the operation hitherto practised for the removal of the entozoön in this position. As excellent, thanks to Alfred Graefe, as are the results of the extraction of cysticerci from the posterior segment at the bulbus when they are fixed in position—whether subretinal, or in the vitreous adherent to the bulbus wall,—just so unsuccessful are the results when they are movable in the vitreous. In Alfred Graefe's twenty-four operations, seven of the eight failures were in cases of movable cysticerci in the vitreous. The reason for this is evident. When the entozoön is fixed, we can estimate its location with sufficient accuracy, following Alf. Graefe's rules, and make the incision at that point. With a movable cysticercus in the vitreous, an accurate estimation of its location will not guarantee a successful result, since the entozoön, as I found in the case cited above, may change its seat in the course of the operation. On account of these considerations, in closing my communication at that time, I made this proposition for the extraction of free-moving cysticerci from the vitreous:

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[1] From No. 3, vol. xxi. of German edition, 1890.

1. To incise the bulbus at a place convenient for the introduction of instruments regardless of the situation of the cysticercus, and

2. To carry traction instruments into the vitreous with the aid of the ophthalmoscope and so extract the cysticercus.

An opportunity for such an operation presented itself in the autumn of 1886.

On Sept. 27, 1886, a patient came to me with a cysticercus in his left eye. It lay in the unclouded vitreous, and could be diagnosed with certainty at the first glance. There could be seen not only the sharp-contoured circular vesicle having a pronounced hydatid gleam, but in the lower outer quadrant of the vesicle, the fully extended neck with the head, and on the latter four suckers, almost as distinct as in an anatomical preparation. The neck and the head, together with the vesicle wall, were movable, and movements could be recognized even in the suckers.

The cysticercus lay in the upper inner quadrant of the anterior part of the vitreous, and could be seen by lateral illumination; its temporal margin came into view when the eye was turned about 20° upward from the horizontal and slightly to the nasal side; its lower margin became visible when the eye was turned 30° to the nasal side and a few degrees upward. The fundus reflex was perceptible about the vesicle near its margin.

The entire retina was detached, is lower half very deep and irregular; it undulated considerably and seemed opaque and gray-white, while the upper half appeared still somewhat transparent.

The vision was consequently almost entirely lost, the patient having an uncertain perception of light, relatively best upward.

Form and tension of the left eye were normal. The right eye was sound; it was emmetropic and had normal vision.

On repeated examination, the cysticercus was found constantly in the same situation in the vitreous, its head being at times more completely extended, mostly downward and to the nasal side, only once outward; at other times quite drawn in and then recognizable as an intensely white gleaming spot.

On October 8th, for the first time, the head could not be seen. The cysticercus had turned, without changing its location in the vitreous.

On October 12th I performed extraction according to the principles laid down above. The patient did not fear the operation, and agreed that it should be performed with cocaine. This facilitated the execution of the operation ; it could of course also have been made under chloroform narcosis. The patient sat opposite me on a chair. I first made a meridional cut in the lower outer quadrant of the globe, entering a straight lance in the direction toward its centre ; the incision was about 10 *mm* long, beginning 6 *mm* from the margin of the cornea. When the patient, who was well trained from previous repeated examinations, brought the eye into the position in which always hitherto, and just before the incision, the cysticercus had been visible, I did not see it. I found it when the eye was turned straight upward as far as possible, its lower margin then becoming visible. I now passed in an iris-hook, whose movements I could clearly follow ; first to the lower margin of the cysticercus, then above and behind it, while at the same time I turned the point of the hook toward the cysticercus and tried to catch it. *At the first attempt the hook slipped off, the second time the parasite followed and was removed from the bulbus.* The vesicle was not at all, or but slightly, collapsed. On the preparation preserved in pure glycerine one can still recognize to-day the triangular flap wound made by the hook. A small quantity of vitreous escaped from the wound with the entozoön ; this had not prolapsed when the section was finished. The vitreous had not its normal consistence.

After using iced compasses for some minutes, the wound was thoroughly washed out with sublimate 1 : 5,000, then closed by a deep conjunctival suture, and covered with iodoform. The ophthalmoscope used in the operation was fastened to a head-band, and by a ball-and-socket joint was movable in every direction.

The recovery was smooth. On October 26th there was a slight injection of the bulbus, there were four bands in the deeper parts of the cornea, and the pupil was medium wide. The wound was fully cicatrized and smooth, with a bit of pigment healed into its nasal end. The intra-ocular pressure was normal, the eye was not sensitive to the touch. On ophthalmoscopic examination the vitreous appeared quite normal and the path of the hook could not be seen. V = 0.

After his dismissal the patient returned from time to time. On December 9th, almost two months after the operation, the vitreous still appeared clear ; it seemed that the bulbus felt softer than before. Fourteen days later I found, below and outward in the vitreous corresponding to the wound cicatrix, a whitish-yellow vascular membrane of triangular form, with its long side toward the bulbus wall ; a large hemorrhage was below the membrane, and numerous small spots of blood were on the detached retina.

The membrane enlarged little by little ; when last I saw the patient, March 3, 1887, it had invaded the whole breadth of the vitreous.

The eye was not irritated, was nowhere sensitive to pressure, and its tension was slightly diminished ; the anterior chamber was somewhat flattened below and outward ; the pupillary margin corresponding to this part was adherent to the lens capsule, elsewhere free ; the lens was clear. The patient was no more conscious of this eye than of the sound one.

On December 19, 1889, in reply to my inquiry, the patient wrote me : (1) that the right eye was quite sound ; (2) that the pupil of the eye operated on looked gray, excepting a crescentic stripe at its upper margin, which appeared black ; (3) that this eye was a little smaller than the sound one.

In our patient there was a free-moving cysticercus in the nasal half of the vitreous of the left eye ; it was not adherent to the bulbus wall. The cysticercus just before the operation lay in the upper-inner anterior quadrant of the vitreous, and immediately after the ending of the section it lay in the upper half of the vitreous, but so far forward that only its lower margin could be seen. There can be no doubt but that, under such circumstances, *without the aid of the ophthalmoscope*, the operator is at the mercy of blind chance, and that in the majority of cases the extraction of a movable cysticercus from the vitreous fails. On the other hand, the chances of success in the operation are certainly not unfavorable if the interior of the eye is illuminated by the ophthalmoscope. The problem which the operator has to solve consists in the extraction of a vesicle about 4 *mm* in diameter from a spherical body about 17 *mm* in diameter, with almost fluid contents. I believe, then, that the extraction of a cysticercus movable in the vitreous will succeed in most cases, or at least more frequently than heretofore, if the operator makes use of the ophthalmoscope in the operation.

A second question, none the less important, is naturally *how the vitreous and the eye in general tolerate this interference.* I shall merely remark, regarding this point, that in my case the result fulfilled all reasonable demands: the operated eye held its form; it did not become phthisical; it did not arouse sympathetic inflammation in the second eye. In order to preclude all doubt as to the points just mentioned, I have waited three years with my publication. The present condition may surely be regarded as stationary. Since the eye, before the operation, was almost blind from amotio retinæ, nothing can be concluded from my case respecting this point. The course of this case was peculiar, in that for two months following the operation the vitreous remained clear, so that the path of the hook inside the eye could not once be seen. Only after this length of time was a yellow vascular membrane developed, starting from the cicatrix and gradually invading the entire breadth of the vitreous chamber. While the cause of the development of this inflammation does not appear clear to me, I should assume that it need not be sought in the escape of contents of the entozoön. If the latter had been irritating to the vitreous, the effect must have shown itself soon after the operation. My case seems to show that there should be no objections on this ground to my procedure. Furthermore, the conditions were here especially unfavorable, as the parasite lay far upward and the instrument therefore had to describe a long course in the vitreous.

I selected as a *traction instrument* a strong iris hook made for this special purpose; I had also fine straight iris forceps, made with sufficiently long branches, in case the hook tore through the cysticercus wall. The hook is to be preferred, as it disturbs the vitreous structures much less than forceps, which must be carried in closed, opened within the vitreous, and again closed.

In incising the bulbus, I use a lance, paying attention to the distant seat of the entozoön, in order with a sharp instrument to divide the vitreous structure in the direction in which the traction instrument was to be introduced. I here followed an idea, suggested by Hirschberg, if I remember rightly, for the extraction of pieces of steel from the interior of the eye by the electro-magnet. When one can reach the cysticercus by a shorter path than in my case, the cutting through the bulbus wall with a Graefe linear knife would involve a slighter trauma to the vitreous.

Finally the place where the eyeball should be opened must in the first place be regulated by the position which the bulbus must take, in order that the operator sitting before the patient may see the cysticercus clearly with the ophthalmoscope, and at the same time conveniently reach the cysticercus from the wound. If the operator is right-handed only, the conditions are more favorable, when as in my case the eye is the left.

In conclusion, I must not forget to remark that I have been and am now aware that the operation under consideration is a severe one. Its danger, however, appears to me not disproportionate to the danger of the affection for which it is practised. For this when left to itself ends as a rule in phthisis bulbi, and as Alfred Graefe's statistics show, the operative procedure hitherto practised could not prevent this in most cases.

Whether the ophthalmoscope can be used with advantage in other operations in the vitreous, *e.g.*, in the extraction of foreign bodies, experience alone can determine. It seems strange that hitherto in operations in the posterior segment of the eye, the illumination of the field of operation by the ophthalmoscope had not been tried. A single report of this sort is known to me in the literature. This was by Wecker[1]; he tried with the aid of the ophthalmoscope to pierce a cysticercus lying downward and inward from the disc, using a very narrow Graefe cataract-knife. The operation failed, since the entozoön escaped the point of the knife. The symptoms of irritation were very slight after this interference.

In connection with the case described above, I shall report briefly a **successful extraction of a subretinal cysticercus,** which I performed in September, 1889.

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[1] "Handbook of Graefe-Saemisch," vol. iv., page 711.

Knapp used it in three cases, for the removal of foreign bodies from the vitreous.—H. K. Published ARCH. OPHTH. AND OTOL., vol. vii., page 337 *et seq.*, 1878.

In a lady, aged thirty-one, having the anterior segment of the globe normal, the parasite was recognized through dense membranes in the vitreous, when the patient turned the eye as far upward as possible and at the same time a little outward. The head could be recognized as a gleaming white spot seldom moving; over the vesicle retinal vessels were clearly seen. When the eye was turned 45° outward, the temporal margin of the entozoön came into view, and when turned 10° outward, the nasal margin, both sharply defined. The nasal half of the retina lay on the choroid, the temporal was detached; there was a paracentral scotoma 20° in extent.

The vision was = fingers at three metres, apparently with central fixation, not improved by glasses.

On September 24th the cysticercus was extracted in the following manner: I first marked on the cocainized eye the vertical and horizontal meridians near the upper and outer margins of the cornea, by small superficial cuts in the conjunctiva bulbi. The patient was then deeply chloroformed. While the assistant rotated the bulbus downward with forceps fixed at the upper margin of the cornea, I cut the bulbus in the direction of the bisecting line of the upper half of the upper outer quadrant, from outward inward, layerwise, first the conjunctiva, then the capsule of Tenon, and, after completely checking the hemorrhage, the sclera. As the scleral wound gaped only in its middle portion, and not at the angles, I divided these parts with straight scissors, first toward the æquator, then toward the cornea. *The cysticercus escaped of itself immediately after the second cut of the scissors, without the loss of a drop of vitreous from the wound.* The wound was about 10 *mm* long, and began about 6 *mm* from the corneal margin.

After the use of iced compresses, the wound was washed out with sublimate 1:5000, the conjunctival wound closed by three sutures, dusted with iodoform, and both eyes bandaged.

The healing went on without accident.

On testing the vision accurately, ten days after the operation, the patient complained of a dark spot in the middle of the field, fingers were barely recognized at two metres' distance by eccentric fixation outward. After some weeks, as the vitreous cleared under the use of pilocarpine injections, the cause of the positive central scotoma revealed itself as an intense white sharply bounded detachment of the retina, projecting tent-shaped into the vitreous, beginning at the disc and gradually broadening upward and outward to the periphery, and including the macular region. The remainder of the retina was not detached. Since before the operation the eye had central fixation, and detachment of the retina near the disc and macula was not diagnosed with certainty with the ophthalmoscope, we must conclude that following, and probably in consequence of, the operation, a retinal detachment had developed, or increased, at the macula. A long course of treatment did not improve the condition.

In this patient the cysticercus lay at the upper boundary of the opthalmoscopic field. In estimating the position for the wound, I assumed that the anterior boundary of the part of the fundus visible with the ophthalmoscope lay a little anterior to the æquator, for the reason that the fundus can be seen for a short distance only—2–3 disc diameters—anterior to the points of exit of the venæ vorticosæ. The venæ vorticosæ, however, according to the painstaking investigations of Fuchs,[1] enter the inner surface of the sclera 2½–3½ *mm* posterior to the æquator. Since now in a schematic eye 24 *mm* long the æquator is distant, following the chord, about 12 *mm* from the corneal margin, as direct measurement on Graefe's schema[2] shows, I had estimated that the middle of the parasite lay about 12 *mm* from the corneal margin. Meanwhile the very accurate computations and observations of Groenouw,[3] touching the question of the anterior boundary of the ophthalmoscopic field, have been published in the last number of *Graefe's Archives.*

Groenouw came to the conclusion that the boundary sought lay 8.5 *mm* from the margin of the cornea. In confirmation of his investigations, he gives an observation of Laqueur's,[4] who recognized with the ophthalmoscope the anterior end of a scleral wound, made with a lance for a magnet extraction, ending 10 *mm* from the corneal margin.

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[1] *v. Graefe's Arch. f. Ophth.*, vol. xxx., 4, p. 23.

[2] *L. c.*, vol. xxiv., 1, p. 212.

[3] *L. c.*, vol. xxxv., 3, p. 29.

[4] *Centralbl. f. Augenheilk.*, 1888, p. 291.

In the question under consideration, the second of the four cysticercus operations, which I reported in 1885, is of value. The posterior end of the scleral wound lay 11–12 *mm* from the margin of the cornea, and was seen with the ophthalmoscope for a distance of one disc diameter, on the boundary of the visible fundus; the latter lay therefore 9.5–10.5 *mm* from the margin of the cornea. This case becomes the more valuable for our question from the fact that the Graefe knife was passed perpendicularly through the sclera, and consequently the inner wound corresponded accurately to the outer. I should conclude from the observation of Laqueur, and from my own, that the possible error of Groenouw's computation, which he estimates as 1 *mm*, should be counted rather toward the positive than toward the negative side.

A CASE OF METASTATIC CARCINOMA OF THE CHOROID.

BY DR. S. SCHULTZE, OF FRANKFURT-O-O.,
LATE ASSISTANT AT THE UNIVERSITY EYE CLINIC IN BERLIN.

(*With plate ix. of vol. xxi., Germ. ed.*)

Translated by H. KNAPP.[1]

WHILE it is well known that primary tumors of the choroid have often been observed, metastatic ones are very rare. So far only four cases of the latter have been laid down in literature, and they all were metastatic carcinomata, which developed rapidly and were soon followed by detachment of the retina and complete amaurosis.

The first case is described by Perls (*Virchow's Archiv*, vol. lvi., p. 437). The following condition was found at the autopsy of a person who had died of primary carcinoma of the lungs:

In the left eye: retina detached on inner and lower sides. Along the whole extent of the posterior half of the eyeball the choroid enlarged to 2 *mm* in thickness, almost as hard as cartilage, forming, near the surface, round opaque, white, slightly prominent and uneven tumors, of which almost all had a somewhat depressed centre. Right eye: the retina adheres to the choroid. In the choroid there are also several infiltrations, slightly prominent, about the size of a bean, similar to those of the left eye. A certain number of the infiltrations are situated in the deeper layers of the choroid, covered in many cases by a thin, vascular, brownish layer. Several single and quite small infiltrations, of 0.5 to 1 *mm* in diameter, entirely white on their surface, lie upon the pigment layer. The larger infiltrations reach to the sclera, and are, in part, firmly connected, with or without, however, penetrating into it.

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[1] From vol. xxi., No 3, Germ. ed. 1890.

On sections the infiltrations are opaque, yellowish-white, dry, partly friable, and, especially in the larger ones, closely packed, yellowish plugs are noticed embedded in a bluish-white, prominent, and harder substance. Under the microscope numerous canroid pearls are found in all infiltrations, freely anastomosing with one another, separated by a scant, fibrillar matrix, which contains, here and there, oblong nuclei and oval, spindle-shaped cells, as well as deeply colored corpuscles of the same structure as the normal pigment cells of the deeper layers of the choroid. In the capillary layer of the choroid, near the infiltrations, were found more or less enlarged capillaries filled with carcinoma cells, consequently an embolism or implantation of carcinoma cells.

The second case was presented in November, 1882, by J. Hirschberg at the Berlin Medical Society. It referred to a woman, aged fifty-two years, who had been suffering nine years from carcinoma of the right breast. The disturbance of vision in the right eye existed six months, that in the left two months.

Through the ophthalmoscope could be seen on both sides a flat, cake-like, hard thickening of the choroid, by which the retina was raised. The neoplasm showed itself first above the optic nerve, later also below it ; its height was over 2 *mm.* The rapid growth of the tumor as well as the constant diminution of vision could be observed until her death, which occurred in the spring of 1883. An autopsy was unfortunately not permitted. (Eulenburg's *Realencyclopädie*, vol. xiii.)

The third case was also presented in November, 1882, before the Berlin Medical Society by Schöler. A few months later, after the death of the woman, aged thirty-three years, who for six years had been suffering with cancer in the left breast, the eyeballs and microscopic experiences were demonstrated by Dr. Ulthoff in the Berlin Medical Society. Carcinomatous degeneration of the choroid was found in both eyes and larger retrobulbar carcinoma nodes in the retina of one eye.

The fourth case was communicated by J. Hirschberg, (Eulenburg's *Realencyclopädie*, xiii.).

In February, 1884, a woman of twenty-eight years, was examined,who had had her right breast removed on account of carcinoma in 1884. She said her left eye had become blind a short time ago. Ophthalmoscopically a dark greenish-gray vesicle, resembling detachment of the retina, was discovered in the lower half of the globe; perception of light absent, tension diminished. The right eye was normal. End of February death occurred. The hardened eyeball showed in the section a large tumor of the choroid, adjoining the sclerotic, protruding into the interior of the eye with its smooth, very convex, anterior surface, terminating toward the front in the equatorical region with a sharp, round edge toward the back, and covering the entrance of the optic nerve. An encroachment upon the sclera could nowhere be ascertained. The microscopic examination showed an epithelial carcinoma with extensive hemorrhages. The retina was raised in the shape of a funnel, and highly degenerated.[1]

To the four above described cases the following may be added. This case came under observation in the private clinic of Schweigger, in Berlin, and was very kindly given over to me for description.

Mrs. X., thirty-four years old, came for the first time to the consulting-room. She stated to have noticed, for about three months, a diminution of sight in the left eye, accompanied latterly with lachrymation and pain.

The pain radiated over the forehead and face (in the region of the trigeminus), and occasionally was increased to severe attacks. The external aspect and tension of both eyes were normal. Left with $-\frac{1}{60}$, $S = \frac{5}{6}$, right $S = \frac{5}{5}$. Ophthalmoscopically a detachment was discovered in the upper-outer part of the left retina, which extended toward the papilla as far as two disc-diameters, with projecting sharp boundaries. Nothing like a tumor could be seen through the detached retina, nor could a rupture of the retina be discovered. A defect existed in the inner-lower part of the visual field. The probable diagnosis was *tumor intraocularis*. On further inquiry it was found that a carcinoma of the right breast and swelling of the glands in the right axilla were noticed in July, 1887, and that on the 5th of December, 1887, v. Bergmann amputated the right mamma and removed the (carcinomatous) lymphatic glands of the right armpit.

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[1] A. SCHAPRINGER, of New York, published another "Case of Metastatic Carcinoma of the Choroid," in N. Y. *Med. Presse*, Sept. 18, '88, and N. Y. *Med. Journal*, '89, p. 109. (See an abstract of the case in THESE ARCHIVES, vol. xviii., 1889, p. 228.—H. K.)

When the patient came again on September 3, 1888, a complete detachment of the retina was found in the left eye. S = 0. Pupil large and without reaction. Under the upper-inner part of the conjunctiva there was a flat, reddish swelling, having the appearance of an episcleritis. For some time the pain in the eye had been diminishing ; on the other hand, the patient complained of severe pain in her whole left side.

The left eye was removed on September 4, 1888. A few days later the patient went home with an artificial eye. The pain in the limbs increased, the left arm and leg became atrophic. As nothing could be discovered on the peripheral nerves, a multiple carcinosis inside the spinal canal was assumed. October 12, 1888, the patient died of bulbar paralysis, as I was informed by the attending physician, Dr. Lobeck. No autopsy was made. The right eye had remained perfectly normal.

The enucleated eyeball was placed in Müller's fluid for two months, then washed out, put in alcohol, divided meridionally, embedded in celloidin, and cut in numerous sections.

The sagittal diameter of the hardened eyeball was 24 *mm*, the transverse diameter 22 *mm*. The anterior parts (cornea, anterior chamber, iris, corp. cil., lens, etc.) were normal. The retina was entirely detached and rose from the entrance of the optic nerve on both sides in a direct, slightly undulatory line almost touching the posterior surface of the lens, then curving about 2 *mm* to the right and left it detached itself close to the corp. cil. The vitreous body was thus shrunk to a cone. The space between the retina and sclera was mostly filled by uniformly coagulated, clear exudation. In the upper half of the eyeball there was a flat, cake-shaped choroidal tumor intimately connected with the sclera (see right side of drawing). It began in front about 4 *mm* below the corp. cil., gradually enlarging toward the optic nerve, at which it terminated, overlapping at the entrance with a round border. Its internal surface was nodular and as black as the rest of the choroid. The retina was closely united to the lower edge of the tumor to the extent of about 2 *mm*. The tumor measured at its base 19 *mm* by 16 *mm*, and was 5 *mm* thick. Laterally and anteriorly gradually the tumor merged into the choroid. The color of the tumor was brownish-green on the surface, shading into black-green toward the periphery. In the anterior third of the tumor the sclera was very much thinned, relaxed, and pervaded with dark masses of the tumor. In the posterior, inferior part of the eyeball (left side of the drawing), 8 *mm* above the entrance of the optic nerve, separated from the large tumor by a space of at least 7 *mm* in width, there was a second, small, flat tumor of the choroid, bean-shaped, and entirely covered by dark pigment, slightly uneven on the upper surface, sharply limited toward the sclera, and everywhere gradually passing into the level of the normal choroid. It was 5 *mm* broad, and 2 *mm* thick. In the section its color was evenly blackish-green. The greenish color was, of course, caused by the chromate of potassa, and had probably been much lighter, more yellowish-gray, when fresh.

Microtome sections were made from all parts of the larger tumor. The smaller tumor was divided into an interrupted series of sections.

The sections of the larger tumor showed the following conditions: The internal surface was completely covered with the normal pigment epithelium and columnar layer of the retina. The club-shaped posterior end of the tumor replaced the lower portion of the optic-nerve fibres, but nowhere the intrusion of tumor elements into the opticus or its sheath could be shown. In its anterior third the base of the tumor was separated from the sclera. In the two posterior thirds the sclera was perforated by tumor masses and considerably thinned. It still clearly showed a small-celled infiltration.

The tumor proper consisted of a delicate meshwork of connective tissue, in the broad interstices of which large cells were accumulated. The cells were irregularly polygonal (Fig. 3), and each contained either one or several large vesicular nuclei, which stained pale with carmin and hematoxylin, often contained distinct dark nuclei and were surrounded by a considerably lighter layer of protoplasm. The size of the cells varied from 10 to 50 μ. The interstitial tissue consisted of delicate anastomosing connective-tissue trabecles, enclosing numerous small, round, spindle-shaped, dark-colored nuclei, as well as a number of black-brown cells, identical with those of the choroid. In parts the interstitial tissue increased in bulk and showed a denser small-celled infiltration and some vessels. On the whole, the tumor had but a few vessels, only where it passed into the normal choroid numerous large vessel lumina were found, engorged with red and white blood corpuscles. Besides these parts, which entirely corresponded to those of a carcinoma simplex, the tumor contained extensive necrotic patches, corresponding to the central, macroscopic, lighter-colored parts, in which the frame-work of connective tissue was distinctly preserved; the cells, however, were unstained and had undergone fatty degeneration. In these necrotic parts repeated hemorrhages had taken place, recent as well as older ones: in the former the single blood corpuscles inside the tissue could be clearly recognized; the latter consisted only of heaps of reddish-brown clusters of hematoidin crystals.

The small tumor is also firmly attached to the sclera; the boundary toward the sclera is, however, sharply defined on all sides, nowhere does the tumor penetrate into its tissue. On both sides the choroid was very much thickened, loosened by the tumor masses, infiltrated with small cells, and in each section the swollen venous blood-vessels were very distinct and numerous. Toward the interior of the eyeball the tumor was covered by the well-preserved pigment epithelium of the retina and basal membrane, beside which almost each section showed from one to three remarkable small flat deposits of the pigment epithelium. These deposits were formed by a granular, non-staining mass containing a few spindle-shaped cells and pigment groups, and were covered by a single layer of spindle-shaped cells. The pigment epithelium of the retina lay under these flat intumescences, which proves that we had not before us the well-known warty excrescences of the lamina vitrosa of the choroid.

The small tumor corresponded in its structure entirely to the larger one, only that the necrotic parts were missing. In the delicate network of fine connective-tissue fibres, which contained pigmented and spindle-shaped cells, typical cancerous cell-nests lay closely packed, touching the basal membrane at the top and the sclera at the bottom, at times pushing at some parts into the relaxed choroid (Fig. 2). From the two hundred sections into which this tumor was cut, about twenty successive ones showed an interesting secondary condition. Between the lamellæ of the sclera a large vein passed diagonally from above downward, in some sections almost below the whole base of the tumor, *i.e.*, at a length of about 3 *mm* (comp. Fig. 2). It had normally thin walls, and while passing through the sclera its normally wide lumen was filled with closely packed red and scant white blood corpuscles; only for a short distance, about in the middle, the contents were more granular and showed a distinct net of fibrine. Above the sclera the lumen gradually enlarged, so that the tumor seemed to have been pressed upwards. The vessel terminated in the tumor with a pear-shaped enlargement (Fig. 2), ran in another direction, and thus left the plane of the section. The club-shaped swelling of the vein in the region of the tumor was filled with a thrombus, consisting of the same magni-nucleated epithelium cells of which the tumor was composed, and the centres of which had already undergone granular degeneration. The thrombus was tightly fastened to the superior vascular wall, which was distinctly thickened, and in a number of sections it could be clearly seen how the vascular wall was perforated by the tumor; tumor and thrombus there merged; the tumor had penetrated into the vessel. At this place the tumor itself had no longer its largest dimensions, being about 4 *mm* in breadth and 1.5 *mm* in height. A broad space on the whole lower and anterior sides, between throm-

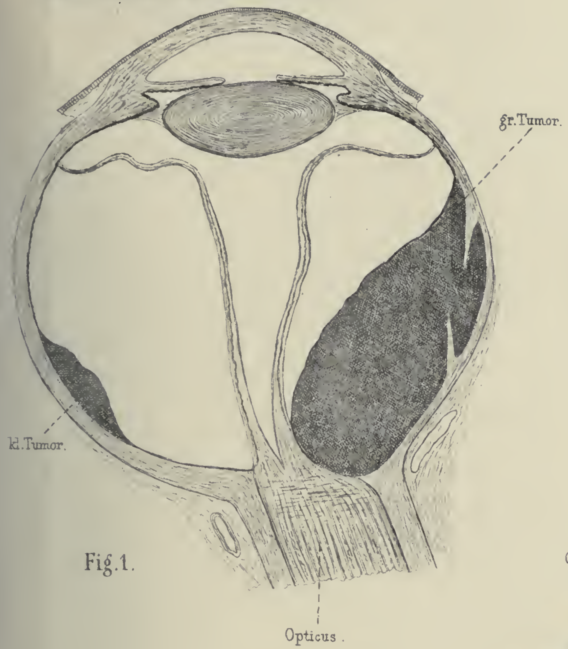


Fig. 1.

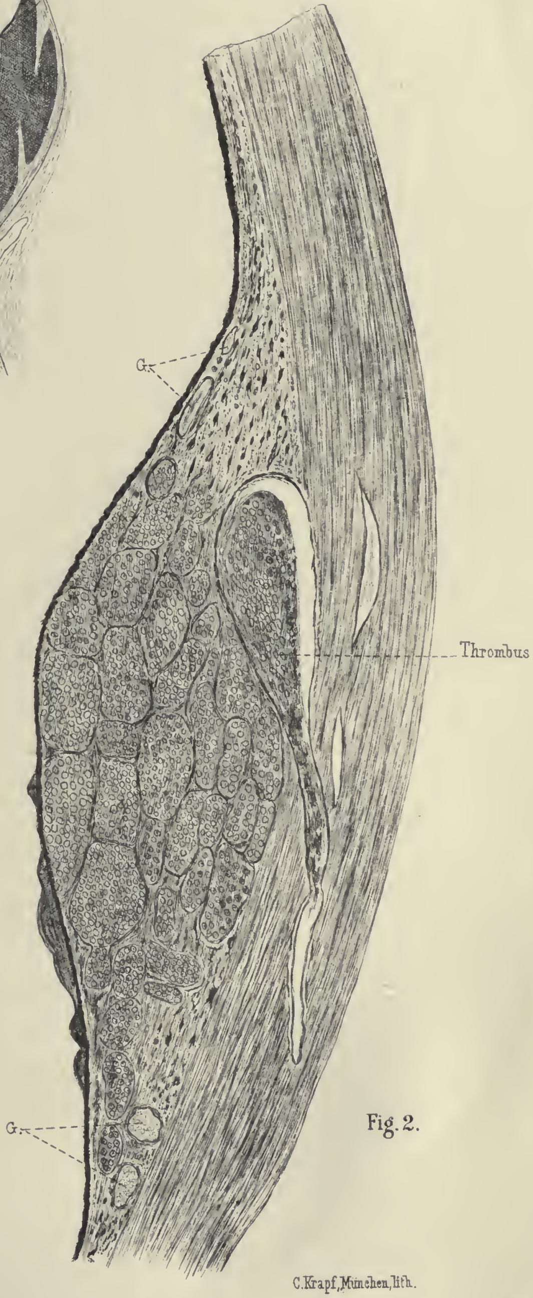
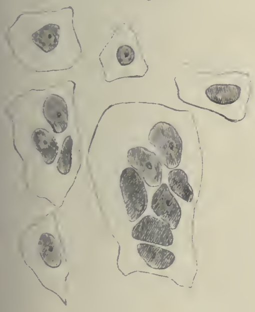


Fig. 2.

Fig. 3.



Dr Schultze, del. C. Krapf, München, lith.

bus and vascular wall, contained some normal red and white blood corpuscles, a proof that the circulation had not entirely ceased; the thrombus was therefore parietal, not yet obstructing.

By the above description it is clearly shown that the two tumors are metastatic carcinomata of the choroid; also the history of the case, especially the rapid growth and the pain, would indicate it. Sarcomata of the choroid—only such could come into question—grow slowly and very rarely cause such intense pain. The small tumor being cut into sections, from one end to the other the larger one examined in various regions and directions, showed everywhere the same definite picture.

The just-described case is very similar to the second of Professor Hirschberg, with this difference, however, that it broke through the sclera. Common to all the cases is the flat expansion of the pseudoplasm, their width always considerably exceeding their height.

THREE CASES OF BONY ORBITAL TUMORS.

By Prof. ADAMÜK of Kasan.

Translated by Dr. W. F. Smith, Chicago, Ill.[1]

IN Nos. 1 and 2 of vol. xx. of these ARCHIVES, appeared an article by W. F. Smith, entited "Case of Diffuse Hyperostosis of the Greater Wing of the Sphenoid—Right Side." In concluding this article the author states his deductions as to the etiology of the neoplasms in question, and in consideration of the materials at present available, he arrives at the following conclusion as to the character of bony neoplasms of the orbit; "Osseous tumors of the orbit, therefore, naturally divide themselves into two classes by the following points of difference: 1st, exostoses and hyperostoses originate from injury of periosteum or bone; and 2d, encapsulated osteomas have a fœtal origin and appear with the development of the bone cavities or sinuses, not later than the thirtieth year; whereby the last-named tumors constitute, as the author declares, the so-called *ossa eburnea*, viz.: are ivory-hard and enveloped by mucous membrane, while the first consist of true bone and appear to be covered with periosteum. "These well defined characteristics," Smith continues, "seem to make the two classes of orbital tumors sufficiently distinct," etc.

Now whether the deduced conclusions are absolutely correct or not can only be determined, in my opinion, by a large number of observations. From the fact that such cases are met with so rarely that the number at present reported does not exceed ten, the following account of three cases of neoplasms of this kind, selected from among my own observations, will be of interest, especially as they seem to be partly in accordance with and partly against the conclusions of W. F. Smith.

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[1] From vol. xxi., No. 3, Germ. ed., 1890.

The FIRST of these cases concerns a twenty-two-year old young man who came under my observation in 1870, almost twenty years ago. The patient, who had journeyed from Astrachan, presented, on his reception to our clinic, a remarkable abnormality of his right eye, which was strongly crowded forwards and at the same time seemed dislocated outwards.

The upper lid, though it appeared somewhat tense, failed to cover the bulb. On palpation we felt an uneven, immovable, hard tumor on the upper orbital wall filling almost two thirds of the orbit, movements of the globe normal, pupil responsive, S= 0.5, venous congestion of the retina. The hard tumor, which the patient had felt in the inner-upper corner of the orbit, had grown more rapidly during the last two years. In view of the extreme hardness of the neoplasm and its intimate connection with the bony wall of the orbit it was not difficult to diagnose an exostosis of the upper orbital wall. In consideration of the fact that the vision of the affected eye was still good and that the latter could not fail to be affected by the continued irritation due to the decided dislocation; further, in accordance with the pressing desire of the patient, I determined to remove the neoplasm by an operation. The nodulated form of the tumor, which seemed to indicate a certain isolated condition of the same, as well as the youth of the patient, gave hopes that I should meet with no particular difficulty in the operation, as I had lately seen a similar case in Paris, where the removal of such a tumor, from a nineteen-year old patient, had been successfully and easily accomplished by Professor Richet, after simply cutting through the soft parts with a pair of bone forceps. It resulted quite otherwise. After anæsthesia by chloroform had been secured an incision was made along the shaved eyebrow and was continued deep enough to expose the neoplasm. Separating the surrounding soft tissues I seized the tumor with the bone forceps, after the example of Richet, and tried to loosen it from its location. But this proved quite impracticable; for while the assistants held the patient's head so firmly as to prevent any rotation, the tumor remained absolutely immovable. Under these circumstances the contemplated removal of the tumor was only possible by means of the bone saw. At first we used the cock's-comb saw, but to reach the deeper parts other apparatus was found necessary. In this the best service was rendered by the Heine osteotome, by the aid of which we finally succeeded in reaching the deeper parts of the neoplasm. This required two hours of work, during which two osteotomes were broken, so dense was the bone sawed through. On account of the depth of the posterior surface of the tumor it could not be reached by the saw, and the remaining part had to be broken with the bone forceps. The neoplasm showed ivory hardness such that no chisel could make an impression on it. The neoplasm measured 39 *mm* by 35 *mm* by 19 *mm*, and weighed 23 *grm.*

After the operation the healing was prompt and normal in its course. The lips of the wound, united by sutures, closed for the most part, per primam. The bulb assumed its original, normal position and retained its functions perfectly. By the end of four weeks there remained only a small opening in the wound leading to the sawed surface, a slight ptosis and a certain obstruction to upward movement. The ophthalmoscopic appearances were entirely normal. In this condition, the patient returned to his home. His later history remains unknown to us.

From the above data it follows that, in this case, we have to do with a neoplasm of fœtal origin, the point of origin of which was to be found in the neighborhood of the frontal sinus, and which therefore may be considered a so-called encapsulated osteoma. To what extent it was covered with mucous membrane rather than with periosteum I cannot say. I remember, however, very well that sawing through the neoplasm was preceded by the tearing of an enveloping membrane. Concerning the character of this latter I can say nothing since it was allowed to remain *in situ*. Undoubtedly, however, it could not have been considered an isolated or encapsulated tumor ; on the contrary, the osteoma was intimately connected with, and formed a part of, the neighboring bone. It is worthy of remark that the ablation of the neoplasm was successful, in spite of the anatomical construction being so very unfavorable to operative methods for its removal.

The SECOND of my observations concerned a bony, orbital tumor in a man thirty years of age. This case came into our clinic two years after the first. The tumor was much smaller, not larger than a hazel-nut, was situated in the upper and inner angle of the orbit, and its true character manifested by its complete immobility and remarkable hardness. The patient stated that two years before he had first noticed something uneven in the angle of the eye, and that the tumor had gradually increased in size since then. There had been no pain. The patient could assign no cause. Syphilis was not present. The eye was only slightly dislocated. The most remarkable symptom, however, consisted in a decided ptosis, as well as an almost complete lack of movement of the bulb upwards, whilst all other functions of the N. oculomotorius were unaffected. The further history of this case is unknown, as the patient absolutely refused to submit to an operation.

After some time I had the opportunity of observing a THIRD case of orbital osteoma, which occurred in a man thirty-five years of age and which had already reached considerable dimensions. On his entrance we found a large and painful swelling, and an ectropium of the eyelids, between which a pus-covered mass, the remnants of the eyeball, came to view. Upon explaining to the patient that he could expect no relief from pain except by an operation, he assented ; chloroform was administered, and the remnants of the bulb were removed. Continuing the anæsthesia, I proceeded to examine the orbital cavity, and it was not difficult to determine that almost the entire orbit was occupied by a tumor, extending from the upper orbital wall downward, and leaving a slit, only a few millimetres in width, between the tumor and the lower wall of the orbit. This narrow slit gave exit to the compressed optic nerve and the ocular muscles. Since nothing could be gained by the removal of the tumor, I determined to leave the parts as they were, and accordingly closed the wound by a tarsoraphy. The union of the lids was completed by normal processes, and in place of the unsightly mass that existed before there now remained an even surface of cuticle slightly elevated above the surrounding parts, but not at all sensitive to pressure, and the patient was dismissed in good condition at the end of four weeks.

In connection with the diagnosis of orbital osteoma the statements of the patient acquired a particular signification. When relieved of his pain by means of the operation, he informed us that he had remarked the development of a bony protuberance at the inner angle of the eye ten years before, without there having been any visible cause, such as a wound, or the like. For some time the vision was unaffected. With the development of the tumor the eye was forced out of the orbit, although he could still see. There was great lachrymation, and the eye became red and painful. Finally the eye became opaque, and panophthalmitis ensued. From this time it is evident that, in the beginning, an ordinary exostosis of a fœtal origin had developed.

We have, therefore, before us a case of exostosis, undoubtedly of fœtal origin, but which did not present itself as an entirely isolated or encapsulated tumor, but, on the contrary, as an ivory-hard, indurated tumor, which invaded the entire upper orbital wall. One thing common to all these cases is undoubtedly correct—that is, the development of the neoplasm took place before the thirtieth year; further, the absence of any possible traumatism, and finally, the undoubtedly early eburneation of the respective osteoma.

In publishing these observations I had only in mind to increase the fund of information regarding these rare cases, with no idea of refuting the conclusions of W. F. Smith.

Since 1878 I have not met with a single additional case of bony orbital tumor, although during this last decade I have examined over twenty-five thousand patients.

A CASE OF LEUCOSARCOMA OF THE IRIS WITH IRITIS SEROSA.

By Dr. PH. LIMBOURG,

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(*With a lithographic plate—tab. xvi. of vol. xxi., German edition.*)

Translated by Charles H. May, M.D., New York.

NON-PIGMENTED sarcomata of the iris are very rare. In the well-known monograph of Fuchs[1] only three cases are mentioned (Lebrun, Dreschfeld, Knapp), and these are contrasted with thirteen cases of melanosarcoma. Subsequently leucosarcomata were described by Sauer[2] and by Zellweger.[3] Melanosarcomata, on the other hand, have been reported so frequently that the ratio expressed by Fuchs has been preserved. Another case of leucosarcoma was observed in Prof. Laqueur's clinic; this case was peculiar in having serous iritis associated with it:

A girl, seven and one half years old, was admitted to the clinic on account of partial loss of sight in the left eye, which had lasted three weeks. Examination showed the following: Vision in the affected eye decreased to the perception of fingers at one metre. Slight episcleral injection. Numerous fine deposits on the posterior surface of the cornea. Anterior chamber increased in depth. A new growth of yellowish color and smooth surface occupied the entire upper and inner quadrant of the iris and extended to the cornea. With the lens, tortuous vessels are seen at the upper margin of the tumor. Tension of the eyeball somewhat increased ($+T_1$). The pupil was irregular; after instillation of atropine, it enlarged and became obliquely oval. The deeper portions of the eye could not be examined. Right eye normal, excepting old opacities.

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[1] "Das Sarcom des Uvealtractus," Wien, 1882.

[2] Inaug. Dissert., Halle, 1883.

[3] *Klin. Monatsbl. f. Augenheilk.*, 1888, Bd. xxvi., S. 366.

As there was no reason to suspect syphilis, the diagnosis lay between solitary tubercle and sarcoma. The family history supported the diagnosis of tubercular new growth. The father and two older children had died from tuberculosis; the patient was a delicate, scrofulous girl who had had rhachitis for a long time. This diagnosis received additional support from the absence of pigmentation—for sarcomata of the uveal tract are usually melanotic. The occurrence and the rapidity of growth of the tumor could not be ascertained on account of the youth of the patient. The fact of the tumor having been observed only two weeks could be attributed to the occurrence of serous iritis, since occasionally diseased or weakened eyes become "infected." Neither was the age of the patient decisive, for sarcomata are also observed in young persons. Against the diagnosis of tuberculosis were the dilatation of the pupils after atropine and the absence of posterior synechiæ.

Iridectomy, recommended by Graefe[1] and Mooren,[2] and which has been done successfully in a number of cases of tumors of the iris which were well circumscribed, was not considered in this case, on account of the size of the tumor and the inflammatory manifestations; the eye was enucleated by Prof. Laqueur.

The eyeball was then examined, an incision being made through the centre of the tumor. The latter measured 5 *mm* in its longest diameter and 1.5 *mm* in thickness. The incision showed it to be of an almost pure white color, and it seemed limited to the iris. Anteriorly it extended to the cornea, with which, possibly, it had become loosely adherent in the angle. The divided eyeball was hardened in Müller's fluid and in alcohol.

Upon microscopical examination, Professor v. Recklinghausen pronounced the tumor a *non-pigmented sarcoma*, formed of spindle-cells, between which were a few small round-cells; there was a small amount of intercellular fibrous tissue; there were few blood-vessels. Fatty degeneration in many portions of the tumor (Fig. 1, *a*). The anterior and posterior surfaces were bounded by pigmented iris-tissue. The new growth extended farther than was apparent on microscopical examination ; it involved the adjoining portions of the ciliary body, extending to the commencement of the ciliary muscle, and encircled the canal of Schlemm. The greater portion of the iris, which seemed normal, was also found to contain numerous areas ; thus masses of the tumor were found extending somewhat into the pupil and arising from the opposite side of the pupillary margin from the main portion of the new growth and at the anterior surface of the iris (Fig. 1, *b*). These masses were very limited in certain sections, but in others they revealed a direct connection with growths which had caused extensive destruction of the pigment layer in the posterior portion of the iris (Fig. 1, *c*). Sarcomatous masses, which projected considerably into the lower iris angle, were especially extensive (Fig. 1, *d*). Whilst the eyeball was *in situ*, these masses were not visible—partly because they were hidden by deposits upon the cornea and partly because the observation of this portion of the anterior chamber is difficult in itself. In making sections of the globe imbedded in celloidin, this projection gave rise to the impression of being due to the serous iritis, and possibly consisting of a loosened collection of corneal deposits. Upon microscopical examination, this mass, the transverse section of which was round, presented the structure of a new growth. Here and there it appeared only slightly adherent to the iris ; but often sarcomatous masses extended from it deeply into the iris and ciliary body. Of the latter the connective-tissue boundary layer exclusively and the ciliary processes were involved. Sarcomatous areas could be demonstrated extending to the centre of the iris (Fig. 1, *e*). The *pars ciliaris retinæ* was destroyed by it in many places. The ciliary muscle was entirely free from involvement.

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[1] *Arch. f. Ophth.*, 1861, Bd. viii., 1, S. 288.

[2] "Ophthalmiatrische Beobachtungen," Berlin, 1867, S. 127.

Thus an extensive infiltration of the rest of the iris had proceeded from the lateral portions of the main part of the tumor. Possibly, certain areas were produced by the separation of small particles from the surface of the tumor, and their subsequent deposition upon different portions of the iris by the aqueous humor.

Cases have been described in which there was partial atrophy of the lens, at times without any change of position, from pressure of slowly growing sarcomata of the iris (Hirschberg, Schiess-Gemuseus) and of the ciliary body.[1] In our case also, several shallow depressions on the anterior surface of the lens could be demonstrated (Fig. 2) ; at these spots, the capsule and epithelium showed no appreciable changes. There were occasional adhesions between the lens and the posterior surface of the iris, opposite sarcomatous areas.

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[1] Fuchs, *l. c.*, S. 216 u. 217.

Our case was of special interest, on account of the existence of serous iritis. Up to the present time Schweigger[1] and Knies[2] have been the only ones to investigate this morbid process anatomically. The changes which I found showed some differences, probably referable to the fact that I was privileged to observe a pretty early stage.

The interlamellar spaces of the cornea were wide and abundantly supplied with cells; but this might vary within normal relations. The form of deposits upon the posterior surface of the cornea was irregular; but in general two varieties could be distinguished. Besides a low, pretty even deposit, there were the well-known larger masses, easily seen microscopically, which are considered especially characteristic in serous iritis (Fig. 3). As already mentioned by Schweigger and Knies, the endothelium of Descemet's membrane had disappeared in part, beneath the deposit. A relationship between the endothelium and the form of deposit could not be demonstrated, although it seemed probable that such a relationship existed. The deposit had the usual composition of detritus and nuclei. Occasionally endothelial cells were seen surrounded by cell-fragments. The deposits were almost completely devoid of pigment; the origin of the slight pigmentation will be referred to again.

The meshes of the ligamentum pectinatum were found enlarged and completely filled with round-cells; this collection of cells extended to Schlemm's canal.

The portion of the iris which was not attacked by the new growth presented an abundant infiltration with round-cells; this infiltration differed from that described by Knies in that I could not find larger collections of round-cells anywhere, and, above all, I could not demonstrate the existence of any arrangement resembling a lymphoma. It extended pretty evenly through the entire stroma, and could be followed into the depressions of the anterior surface of the iris, down to a deposit found here (Fig. 1, *f.*) resembling that on Descemet's membrane, except that there was less structural degeneration. The deposit found over many portions of the surface of the large tumor of the iris was found to consist partly of tumor cells and partly of the products of serous iritis. Excepting destruction produced by the new growth, the pigment layer of the iris was unchanged.

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[1] "Handbuch der Augenheilk.," 5 Aufl., 1885, S. 333.

[2] *Arch. f. Augenheilk.*, Bd. ix., S. 1.

The ciliary body also presented marked cellular infiltration, resembling that described in connection with the iris and like that demonstrated by Knies. In the boundary layer it was difficult to separate tumor from infiltration. The pigment cells of the *pars ciliaris retinæ* were very light in color and deficient in pigment.

The zonula also presented numerous round-cells and pigment-nuclei.

The choroid was apparently normal; the number of round-cells which it contained was probably not increased or only to an insignificant degree.

In addition, there were also deposits on the anterior capsule of the lens, chiefly insignificant in amount; these were found especially in the pupillary area and in the atrophic portions already described. They differ from the deposits on the posterior surface of the cornea and on the anterior surface of the iris in presenting considerable pigmentation; this originated principally in the sarcomatous masses on the posterior surface of the iris and the ciliary body.

Reviewing the important points, our case was one of leucosarcoma of slow growth, occurring in a child, and observed at the beginning of the inflammatory period.[1] The growth had remained unobserved for a long time, since its development had been a slow one, and since the pupil had remained free, and the lens had not become displaced, or at least had not become changed in shape in its central portion. It was not until the occurrence of deposits upon the posterior surface of the cornea and the anterior surface of the lens that the attention of the child and others was directed to the growth, which anatomical investigation showed to be of much longer duration. We cannot even tell approximately what the duration of the first stage was. The increase in tension is easily explained by the partial obstruction of the canal of Schlemm by the tumor.

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[1] H. Knapp, "Die Intraoculutaren Geschwülste," Karlsruhe, 1868, S. 178.

The iridocyclitis serosa, an anatomical examination of which was possible two weeks after its commencement, resulted in a uniform infiltration of the iris and ciliary body. The round-cells escaped from the anterior surface of the iris into the aqueous and formed deposits upon the iris, or were washed against the posterior surface of the cornea forming diffuse or greater and more circumscribed deposits, as a result of which the endothelium of Descemet's membrane was the seat of considerable disturbance. Most of the cells were removed by Fontana's spaces. Pigment was very scanty and in most places it seemed to be due to accidental additions from the new growth.

In conclusion I wish to express my sincere thanks to Prof. Laqueur and Prof. Recklinghausen, for their kind assistance.

Fig. 1.

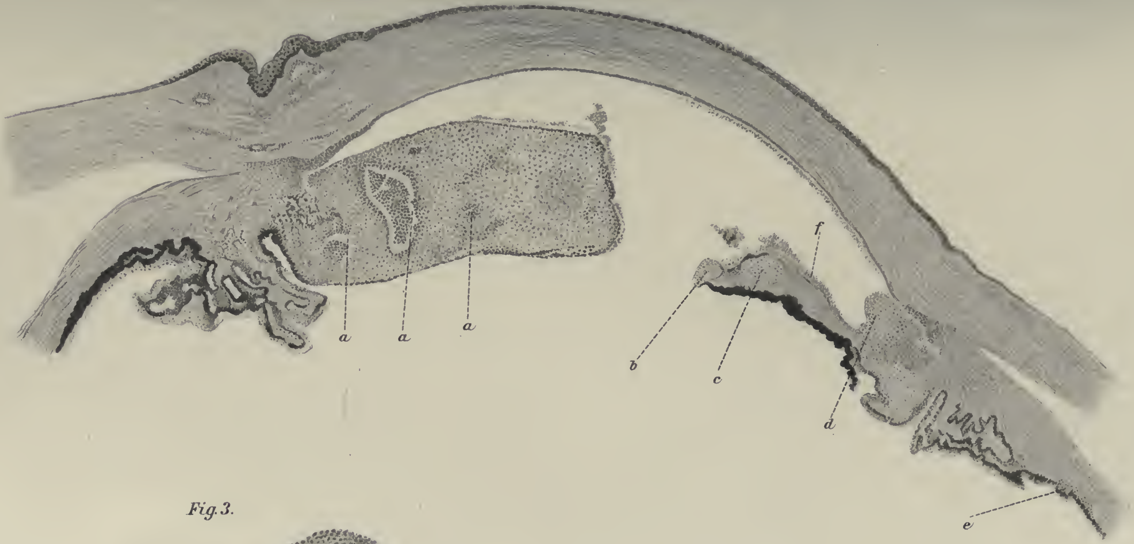


Fig. 3.

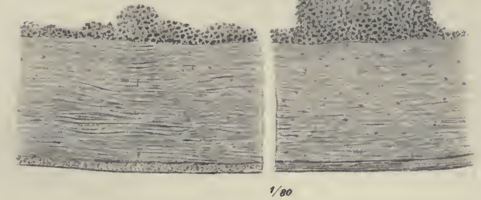
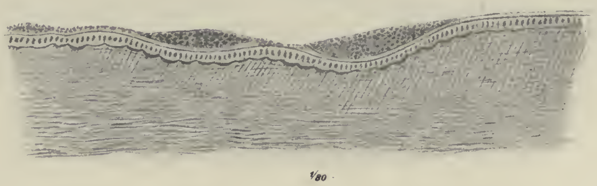


Fig. 2.



E. Kretz, gez.

Lith. C. Kirst, Leipzig.

NODULAR OPACITIES OF THE CORNEA (NODULI CORNEÆ).

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Translated by Dr. J. M. Mills, New York.

(*With a drawing in the text.*)

THE name keratitis punctata is used for the different forms of punctate and nodular opacities of the cornea, whether they are stationary or the manifestation of a recent keratitis. Nearly every author gives it a different construction.

As regards the stationary punctate form of corneal opacities, Graefe[1] incidentally mentions a case which later on will be more minutely considered.

Heymann[2] describes the linear corneal opacities, in which he includes the well-known striped keratitis found after cataract operations, and Raehlmann[3] describes, in detail, similar linear and striped forms of opacities, which sometimes acquire a distant resemblance to the dots or nodules when the thickness produced by the intersection of the different striæ gives the impression of a special formation. He also mentions the punctate opacities which lie in the deeper layers of the centre of the cornea, and which later, through confluence, form opacities, as are often found in the course of a diffuse parenchymatous keratitis; the cornea is generally slightly vascular, iritis is absent. These opacities have nothing in common with the punctate precipitate on the posterior corneal wall observed in iritis serosa.

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[1] *Arch. f. Ophthal.*, Bd. ii., 2, S. 309.
[2] *Klin. Monatsbl. f. Augenheilk.*, Bd. vi., S. 277.
[3] *Klin. Monatsbl. f. Augenheilk.*, Bd. xv., S. 1.

A true punctate keratitis, dependent upon a syphilitic basis, is described by Mauthner.[1] This form is characterized by circumscribed small gray spots in the substantia propria corneæ and in fact in the various layers. The corneal tissue surrounding the focus is only slightly altered. Peri-corneal injection may be absent. The central deposits never attain a larger extension or form an abscess; they can develop quickly and disappear rapidly, leaving no opacities or only the punctate form.

The iris does not participate in this process. This rare disease occurs only in general syphilis, the isolated spots in the cornea are probably small gummata.

Hock and Purtscher[2] have observed a similar disease; the latter reports a case which was complicated with iritis, opacities of the vitreous, and retinitis. He obtained a cure with mercurial inunctions and potassium iodide, as the patient had inherited syphilis.

Schweigger[3] considers the punctate keratitis to be a hybrid variety of the deep corneal inflammation, which is usually complicated with iritis and heals in from two to three months without leaving any traces; it is not infrequently of a syphilitic origin.

Lately Adler[4] has described a disease under the name "keratitis subepithelialis centralis," which Fuchs[5] terms "keratitis superficialis punctata." This disease, according to the reports of both authors, which agree in almost every particular, begins with the symptoms of an acute conjunctivitis with profuse lachrymation, and then, after from eight to twelve days, the changes in the cornea appear, whilst the conjunctival catarrh quickly disappears, and the eye from then on remains unirritated. The corneal opacities consist of small gray disc-shaped spots, scarcely ¼ *mm* in diameter, and of a yellowish-white or gray color; their number varies between ten and one hundred, and they occupy exclusively or principally, at least, the central portion of the cornea, directly under the epithelium or even in the propria. The corneal tissue between them is transparent and shows, sometimes, small, somewhat deeper lying dots. With a strong magnifying glass, the larger spots resolve themselves also into a large number of exceedingly fine small dots. The epithelium does not, by any means, always project above the opacities and is never materially altered. The corneal affection is usually only unilateral, and rarely affects both eyes. The spots gradually fade, and disappear entirely in a few months, at the latest in a year. Complications with iritis or hypopyon have not been observed. This disease is closely allied to "herpes febrilis corneæ," however, it differs from it essentially in the absence of pustular formations on the cornea.

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[1] Zeissl's "Lehrbuch der Syphilis," 5 Aufl., 1888, S. 633.

[2] Un caso di cheratite punctata albescens. *Boll. d'Ocul.*, Bd. vi., No. 11, S. 237.

[3] "Handbuch der Augenheilk.," Berlin, 1880, S. 314.

[4] *Centrabl. f. prakt. Augenheilk.*, 1889. S. 289.

[5] "Lehrbuch der Augenheilk.," Leipzig u. Wien, 1889, S. 189 f. *Wiener klinische Wochenschr.*, 1889, No. 44.

v. Reuss[1] has described the same disease by the name of "keratitis maculosa," but deviates from the description of the other two aforementioned investigators in several points. According to him the corneal spots have a diameter of 1 to 1½ *mm*, are not sharply circumscribed but are encircled by a blurred zone; they are most numerous at the periphery, although the centre is never entirely without them. Their course, according to Reuss, is also somewhat different, as the spots, even after remaining several months, seldom disappear entirely.

The picture, of the disease, as described by Adler and Fuchs, has a certain similarity with the "nodular form of corneal opacities," which, next in the succession of the cases observed, will be more minutely described.

T——, 49 years old, came to the policlinic on June 17, 1886, with a conjunctival catarrh of the left eye, with which he had been suffering eight days. Patient has not, to his knowledge, had any inflammation of the eye in the past few months or years; is likewise ignorant of any cause for his present condition. Careful examination revealed a large number, about fifty to sixty distinct gray opacites in both corneæ, situated close under the epithelium, which in the left eye was somewhat elevated. These opacities occupy the central portion of the cornea, whilst the margin is entirely free; they are of a whitish-gray color and about the size of a millet seed. Later, upon a more exact examination, these spots were found to lie in the superficial (epithelial) layers of the cornea, perhaps in the foremost layer of the corneal tissue proper, whilst between them are scattered a large number of fine points, hardly half the size of the former. Otherwise the corneal tissue between the individual opacities is entirely clear. The individual dots are of a spherical form, sharply circumscribed, and never coalesce into larger spots, but, at the most, form, by juxtaposition, here and there groups of two or three parts. These nodules resemble, principally in size and form, the precipitate on Descemet's membrane in iritis serosa; however, are distinguished from these, apart from the entire absence of any symptoms of iritis, by their distinct subepithelial position, which is made especially manifest by the circumstance that they cause the corneal epithelium to protrude in parts.

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[1] *Wien. klin. Wochensch.*, 1889, No. 34, P. 665.

The conjunctiva of the right eye of the patient was not at all injected, though in the left there was considerable catarrhal injection and a partial defect in the corneal epithelium over the opacities. The iris was entirely normal, also the opthalmoscopic condition, only that the fundus appeared considerably distorted, in consequence of the irregular curvature of the cornea. The treatment consisted, in the first place, in the application of a compressive bandage on the left eye, whilst a ½ % solution of zinc sulphate was instilled, twice daily, into the conjunctival sac of the right eye.

On June 22d the patient was admitted to the hospital, and four days later, after the injection had subsided, we proceeded to scrape off the corneal epithelium of the left eye, and then the opacities were found to be much firmer and more resistant than the epithelium, so that during the abrasion they produced the same sensation as of scraping a grater with a knife. The same operation was repeated, on July 2d, in the second eye, after a previous scarification. Each eye remained bandaged for three days after the scraping, and then a boric acid solution was instilled twice daily.

On July 13th both eyes were without irritation, and the epithelium restored though the opacities did not appear at all changed; at the most they were only somewhat smaller. The vision in each eye was $\frac{20}{100}$ Sn. Patient was discharged.

On Sept. 29th the patient came again to the clinic. Both eyes were slightly injected in the region of the palpebral fissure ; the corneal opacities were entirely unchanged. Vision = $\frac{20}{100}$, which increased to $\frac{20}{50}$ in each eye, after a scraping of both corneæ, made the next day ; however, the appearance of the opacities was not appreciably altered.

Four months later, on February 3, 1887, the patient was again admitted to the clinic. The opacities had increased somewhat on both sides, therefore an energetic scraping was again undertaken. After this the vision again increased to $\frac{20}{50}$, and the gray points in the cornea were not so numerous as before the operation. This same procedure was repeated on November 30th of the same year, and the eyes remained permanently without injection, but the opacities showed no perceptible change, and the vision remained $\frac{20}{50}$. The same condition was still present on May 25, 1889, when the patient appeared for the last time.

In this case the corneal opacities portrayed lasted three years entirely without change. Symptoms of irritation, worth mentioning, were only present in the left eye for the first two weeks, and they appeared to be due to an accidental excoriation of the cornea, as the other eye, affected with the same corneal opacities, was entirely without injection.

The second case, Johanna B——, æt. 17 years, visited the policlinic, for the first time, on July 7, 1887. She had noticed a gradual decrease in the sight for four years, without any symptoms of inflammation. The eyes were found to be without injection, and showed corneal opacities similar to those of the first-mentioned case. S = $\frac{20}{100}$ — 70 Sn. The treatment consisted of the instillation of a solution of zinc sulphate, beginning with one per cent. and increasing to four per cent. ; however, without success.

On December 5th, of the same year, the patient was admitted to the clinic and the following condition noted : Conjunctiva of both eyes unirritated ; in the centre of the cornea, covering a circular surface of 4 *mm* in diameter, and entirely absent in the periphery, were thirty gray, sharply defined, miliary opacities. They were round in form. They appeared as granules in the cornea, and, in fact, in the superficial, epithelial, or sub-epithelial layers ; they projected somewhat above the cornea, which, consequently, appeared to be covered with little humps, still their surfaces were covered with epithelium. And when the cornea was examined with the keratoscope of Placido, the reflection of the black concentric circles appeared, in certain places, as if it was interrupted by distinct interstices that correspond to the small protuberances. With the haploscopic magnifier very fine, dust-like opacities can be seen between the granules, though otherwise the corneal tissue is completely normal. See accompanying figure.

The latter are not shown in the adjacent schematic sketch, which is taken from the history of the case in question. The iris and fundus are normal; only the latter, on account of the corneal opacities, appears considerably veiled and astigmatically distorted. The vision, right, is $\frac{20}{100}$; left, $\frac{20}{200}$, which increased to $\frac{20}{70}$ after scraping the epithelium, though the opacities remained unchanged; at most, the individual nodules did not project so far above the corneal surface as before.

NODULAR FORM OF CORNEAL OPACITIES.

The microscopic examination of the scraped-off epithelium revealed no special histological elements which were characteristic of the opacities. The patient left the clinic on December 18th. She was advised to use, at home, a one-per-cent. solution of zinc sulphate as a mild irritant.

In April, 1888, the patient appeared again at the clinic. The vision was the same as in December, namely, $\frac{20}{70}$ in each eye; the corneal opacities were unaltered. As it is alleged that nævi and corneal leucomas can be removed by the electrolytic treatment, it was attempted with the constant current. The left eye was thoroughly cocainized, and then a small moistened sponge-electrode (kathode) placed on the cornea, whilst the anode lay upon the neck, and then the current up to four elements of a Voltolini dip-battery, gradually slipping in and out, was sent through the eye. For comparison, the right eye was only treated with a twenty-per-cent. zinc solution. After about fifteen sessions up to May 11th, the corneal opacities and vision were absolutely unimproved, quite the reverse, in fact, as a small excoriation of the left cornea was produced by the electricity, necessitating a cessation of the electric treatment for several days. The patient has not appeared since.

Also in this case the corneal opacities remained a year entirely unaltered; very likely, however, they were present four years previous to the beginning of observation.

Reviewing the described picture of the disease it is as follows: With the exception of what was probably an accessory unilateral conjunctival catarrh, in the first case, there was no injection of the conjunctiva of any consequence, nor a vascularization of the cornea, nor an iritic irritation; so that during the whole period of observation it was not a question of a florid process, as a keratitis, but rather a condition of stationary nodular opacities of the cornea. The opacities consisted of sharply outlined gray dots that did not exceed $\frac{1}{4}$ *mm* in diameter and were thickly scattered, did not become confluent, were separated from one another by healthy corneal tissue, entirely free from spots or stripes, only that between these larger opacities lay smaller, almost dust-like, gray dots, which though half as large were more numerous. The opacities occupied almost exclusively the central portion of the cornea and were situated in the superficial layers, but not only in the epithelial layer, which perhaps was affected by them. The larger ones formed small protuberances on the surface of the cornea and disturbed the vision very much, though otherwise the surface of the cornea was smooth; defects of epithelium do not belong to the picture of the disease. At the scraping, the opaque places were found to be firmer and more resistant than the epithelium. They most likely develop very slowly, seem not to overstep a certain limit, and never retrograde spontaneously, at least they remain stationary for years. All treatment is comparatively powerelss; the scraping off of the epithelium produces occasionally, for a short time, a certain amount of improvement of the sight; less successful are the irritant remedies—for example, two to four-per-cent.-zinc solutions; the electric treatment is useless. The name "nodular corneal opacities (noduli corneæ)" appears suitable to characterize the described opacities.

That these opacities originated from an acute keratitis, as perhaps the subepithelial inflammation of Adler, is very doubtful, as only the first patient at the time he appeared for treatment had, for eight days, an irritable condition of one eye. Therefore the opacities must have originated, quite acutely, in the course of a week, which is hardly credible, considering the resistant nature that characterizes them as epithelial or connective-tissue proliferations, not as infiltrations; the supposition is much more justified that the not over-intelligent patient had completely overlooked, as long as he had no pain, the disturbance of sight occasioned by the gradual formation of the opacities (probably continuing through several years), and only sought medical advice when it was accidentally complicated with an acute conjunctival catarrh of one eye.

This is justified by the circumstance that the opacities after three years presented exactly the same appearance as at the stated beginning of the disease. The second patient had no inflammation of the eye at all and only came to the policlinic for treatment on account of the defective sight, produced by the opacities, which was of four years' standing.

The nodular forms of corneal opacities are therefore not a product of an acute keratitis, but rather develop gradually and chronically, without any symptoms of inflammation, and notwithstanding that they continued unchanged for several years, never produced any irritation in the eye. They are most likely, as in the case of Graefe, to be mentioned later, epithelial proliferations, or perhaps also cicatricial tissue. According to the opinion of Professor Förster, with whom the designation "nodular opacities of the cornea" originated, they resemble mostly the moderately prominent corneal spots that are occasionally found in otherwise perfectly healthy corneæ, and which we are inclined to consider proliferating cicatrices, in which at best there is no proof of a previous ulcer.

Of the cases mentioned in the literature, that of v. Graefe (*l. c.*) bears a close resemblance to the described disease.

A patient had acquired an opacity of the cornea of the right eye from an injury produced by a child's finger-nail. A large number of exceedingly fine yellow dots were scattered over the surface of the cornea, between which there were entirely regular transparent spaces. They only occupied the centre of the cornea and were situated directly under Bowman's membrane, or even more externally. As the opacities caused quite a considerable defect of sight Graefe decided to remove them. He succeeded, contrary to his expectations, in easily removing, with the forceps, the entire part of Bowman's membrane containing the dotted opacities. The examination showed that the opacities were situated on the external surface of Bowman's membrane—that is, in the epithelial layer; they formed concentric stratified epithelial globules, whilst the membrane itself exhibited no changes.

These punctate opacities do not entirely accord with those described, as, in the first place, the opacities were the result of a trauma; furthermore, were yellow not gray; and lastly, could be completely detached, together with Bowman's membrane; still, otherwise they possess a great similarity with the "nodular opacities," as also in this case stationary punctate opacities in the epithelial layer of the cornea were present.

Of the other forms of keratitis punctata Mauthner's syphilitic form cannot at all be taken into consideration, nor indeed the linear and striped opacities of Heymann and Raehlmann, whereas the keratitis subepithelialis, according to the description of Fuchs and Adler, as regards the appearance of the opacities, almost completely coincides with the opacities of the cornea just described; it is true only macroscopically, as with the magnifying glass the nodular opacities of the cornea appear as solid structures, whilst the spots of the subepithelial keratitis appear as densely crowded small points.

The important difference, however, to be noticed between the two diseases is, that in the cases in question most assuredly we had not to deal with a cellular infiltration resulting from an acute keratitis. For there could only have been semi-fluid or cellular masses of exudation, which appear, in the course of two to three weeks, in subepithelial corneal inflammation, and then, even though it is after a lapse of months, become smaller, and finally disappear entirely; whilst, on the contrary, the nodular opacities of the cornea constitute small round solid formations, which develop slowly, remain stationary for years, and are much harder than the corneal epithelium, therefore must consist of epithelial proliferations or cicatricial tissue ; whilst cellular infiltrations are softer than the corneal epithelium and, in any case, cannot remain unchanged for years.

Consequently the nodular opacities of the cornea are to be considered a special form of stationary corneal opacities, which, to a certain extent, belong in the same group with the so-called ribbon keratitis. We are not justified in regarding it as a chronic form of the keratitis subepithelialis, or as a sequel of this disease, that, as yet, has not been observed.

NOTES ON BIFOCAL LENSES.

By ARCHIBALD PERCIVAL, M.A., M.B., Camb.,
Newcastle-upon-Tyne.

(*With four figures.*)

I READ with great interest the account of Dr. Gould's improvement in bifocal lenses, which appeared in the December number of the Archives, as I had devised a very similar modification shortly before I saw his paper on the subject. It is possible that the following notes respecting the size, form, and position of the presbyopic segment may be of interest to some readers of the Archives.

The fixation field for near vision need not extend more than 14° on either side of the middle line, therefore the width of the presbyopic segment need not be greater than 2 (k tan 14), where k is the distance of the first principal focus of the eye (*i.e.*, the position of the lens) from the centre of rotation of the globe, 27.4751 *mm*. This brings the width of the presbyopic glass to 13 or 14 *mm*, and is amply sufficient to allow the wearer to read comfortably without moving his head. In my pattern the upper limit of the glass forms a horizontal line as indicated in the sketch. It appears also advisable to decentre this small glass in such a manner that when combined with its larger fellow the optical centre of the two may be traversed by the visual lines in reading. If we assume that a downward rotation of the globes to the extent of 15° takes place in reading, the optical centre of the combination should be k tan 15° or 7.36 *mm* below the horizontal plane, *i.e.*, below the optical centre of the distance glass. Lastly, since a convergence of

3 m. a. occurs when the reading distance is $\frac{1}{3}$ metre, the optical centre of the reading combination should be displaced 2.5 *mm* inwards (if distance between ocular centres 64 *mm*, $\frac{27.475 \times 32}{333.\dot{3} + 13.7} = 2.53$ *mm*).

Now the deviation of a ray which traverses a point V millimetres below the optical centre of the large (distance) lens is $\tan^{-1} \frac{V}{F_1}$, where F_1 is the focal distance of the lens, or $\tan^{-1} \frac{V D_1}{1000}$, where D_1 represents its strength in dioptries. This vertical deviation must be corrected. [1] Let a be the constant representing the displacement downwards of the geometrical centre of the small lens below the optical centre of the reading combination. Then $\tan^{-1} \frac{a D_2}{1000}$ tends to compensate the primary deviation, where D_2 represents the dioptric strength of the small glass, and $\tan^{-1} \frac{V D_1}{1000} - \tan^{-1} \frac{a D_2}{1000}$ is the deviating angle of the prism required to give the requisite vertical focus of the optical centre of the combination. Similarly the prism for the horizontal correction (H) is given by the formula $\tan^{-1} \frac{H D_1}{1000}$. The small prismatic lens (D_2) is cemented to the distance lens in the normal position, *i.e.*, H *mm* inwards, so that the full value of the binocular fixation field at reading distance is obtained, and when the visual lines are parallel symmetrical retinal

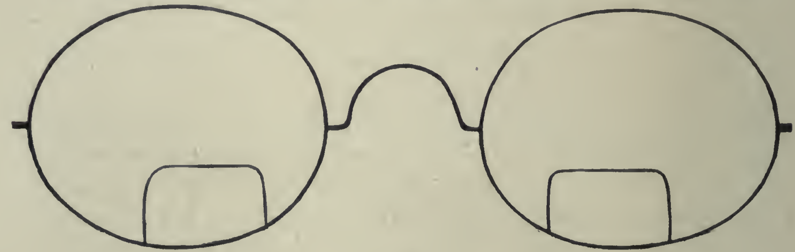

FIG. 1.

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[1] If h_1, h_2 represent the heights of the two lenses in millimetres $a = \frac{h_1 - h_2}{2} - V$. With the dimensions subsequently given $a = \frac{30 - 12}{2} - 7$, or 2 *mm*.

areas are no longer completely covered, hence nearly central (distance) vision may be obtained by one or the other eye.

To take an example: suppose glasses of this kind are to be ordered for a presbyopic hypermetrope H = 2 D, Presb = 3 D. Elliptical plano-convex lenses + 2 D of the usual size, 30 *mm* × 36 *mm* are mounted in the ordinary manner. Prisms are now taken which will compensate for both the horizontal and vertical deviation, their dimensions being 12 *mm* × 14 *mm*. Let φ denote the deviating angle of the prism required for the vertical correction

$$\varphi = \tan^{-1}\frac{\text{V D}_1}{1000} - \tan^{-1}\frac{a\text{ D}_2}{1000} = \tan^{-1}\frac{7.36 \times 2}{1000} - \tan^{-1}\frac{2 \times 3}{1000}$$

$$= \tan^{-1} .01472 - \tan^{-1} .006 \quad = 50'\ 36'' - 20'\ 38'', \textit{ i.e. } 29'\ 58''$$

Let θ denote the prism required for the horizontal adjustment:

$$\theta = \tan^{-1}\frac{\text{H D}_1}{1000} = \tan^{-1}\frac{2.53 \times 2}{1000} = \tan^{-1} .00506 = 17'\ 3''$$

If α be the deviating angle of the resultant prism:[1]

$$\sin\alpha = \sqrt{\sin^2\varphi + \sin^2\theta} = \sqrt{\sin^2 29'\ 58'' + \sin^2 17'\ 3''}$$

$$\therefore \alpha = 34'\ 33''$$

If ρ be the angle of rotation of this prism from the vertical

$$\sin\rho = \frac{\sin\theta}{\sin\alpha} = \frac{\sin 17'\ 3''}{\sin 34'\ 33''}$$

$$\therefore \rho = 29^\circ\ 39'$$

Prisms therefore of deviating angles 34′ 33″ are required set with apices obliquely upward and inward at an angle of 29° 39′ from the vertical of dimensions 12 *mm* × 14 *mm*, and ground convex on one surface to the extent of + 3 D. These are cemented 2.53 *mm* to the nasal sides of the distance lenses. In cases of heterophoria the appropriate prismatic correction can be easily introduced into the calculation.

In the case of presbyopic myopes the base of the correcting prism would be upward, and hence the process known as double-grinding gives more satisfactory and certainly more

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[1] Mr. Ernest Maddox gives these formulæ for resultant prisms in his useful book on the "Clinical Use of Prisms."

graceful results, though there are some attendant disadvantages. In these cases the lower segment forms a meniscus, the optical centre of which lies outside the glass. The vertical upward deviation due to the plano-concave, when the visual lines traverse points V millimetres below the centre of the lens, is $\tan^{-1}\frac{V D_1}{1000}$; the vertical downward deviation due to the convex segment is $\tan^{-1}\frac{V D_2}{1000}$. A prism whose deviating angle is $\tan^{-1}\frac{V D_1}{1000} - \tan^{-1}\frac{V D_2}{1000}$ is cemented on to the plane surface of the plano-concave apex downward, and its free surface then ground to a convexity of $+ 3$ D until the tool has traced out a convex surface reaching to a point about 3 *mm* below the geometrical centre of the large lens. The prism which was used for blocking out the upper segment is then detached, and a lens is formed as in the annexed diagrams, Figs. 1, 2, 3, 4. To take an example, let $D_1 = -7$, $D_2 = +3$, $V = 7$ *mm*. A plano-concave of -7 D is taken, to this is cemented a prism whose deviating angle is $\tan^{-1}\frac{7 \times 7}{1000} - \tan^{-1}\frac{7 \times 3}{1000} = 2^\circ\ 48'\ 20'' - 1^\circ\ 12'\ 20''$ or $1^\circ\ 36'$. This is ground by a 3 D tool in the manner above explained. Unfortunately it is a mechanical impossibility for the tool to trace out a convex surface bounded by a convex margin. It is usually unnecessary to correct

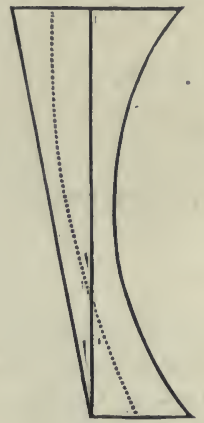

FIG. 2.

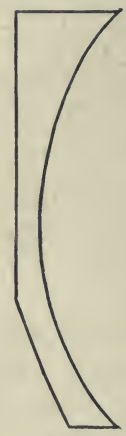

FIG. 3.

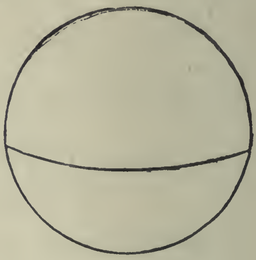

FIG. 4.

the horizontal deviation, as there is frequently slight exophoria in accommodation in high myopia, which a glass of this pattern tends to relieve. Should, however, a correction be necessary, the prism is increased to the requisite degree and the lens rotated to the appropriate position when mounted in the frame. The formulæ for this calculation are given above.

The cost of spectacles made in this way has prevented my ordering them as frequently as I could have wished, but those who have worn them speak of them with the highest praise. I can fully endorse all that Dr. Gould says with reference to his modification, but would add that they are not suitable for astigmatic patients.

It may seem that the formulæ given for prescribing these spectacles are needlessly complex, but it must be remembered that they are designed for those who are doomed to wear glasses always, and a little extra trouble can be well spared in their behalf.[1]

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[1] Messrs. Curry & Paxton, Gt. Portland Street, London, have most skilfully and successfully worked out my designs.

SKIASCOPY: WITH A DESCRIPTION OF AN APPARATUS FOR ITS READY EMPLOYMENT.

By SWAN M. BURNETT, M.D.,

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(*With a wood engraving.*)

SKIASCOPY has now a recognized place among the scientific methods of testing the refraction of the eye, and its employment, while perhaps not general, is increasing in extent. The principles on which it is based are now explained (though not in every case fully or accurately) in several recent text-books on the eye, and in most of the manuals on refraction,[1] so that it is not necessary to consider here the theoretical part of the subject.

While deeming it one of our most valuable means of determining refraction, I still do not hold it superior to all others for all sorts of cases. It has its limits as well as its advantages. Thus, I think that the accommodation of the patient comes in to vitiate the result to a much greater extent than in the direct ophthalmoscopic method. But for children and high degrees of ametropia it is the easiest of any of the methods at present in use, and is as scientifically accurate as any. And, as I have elsewhere[2] remarked, we cannot have at our hands too many means of verifying our diagnosis in refraction.

But while there are few who doubt the scientific ac-

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[1] See paper, by the author, on "Skiascopy," in *Medical News*, Sept. 15, 1888.

[2] Theoretical and Practical Treatise on Astigmatism. Chambers, St. Louis, 1887.

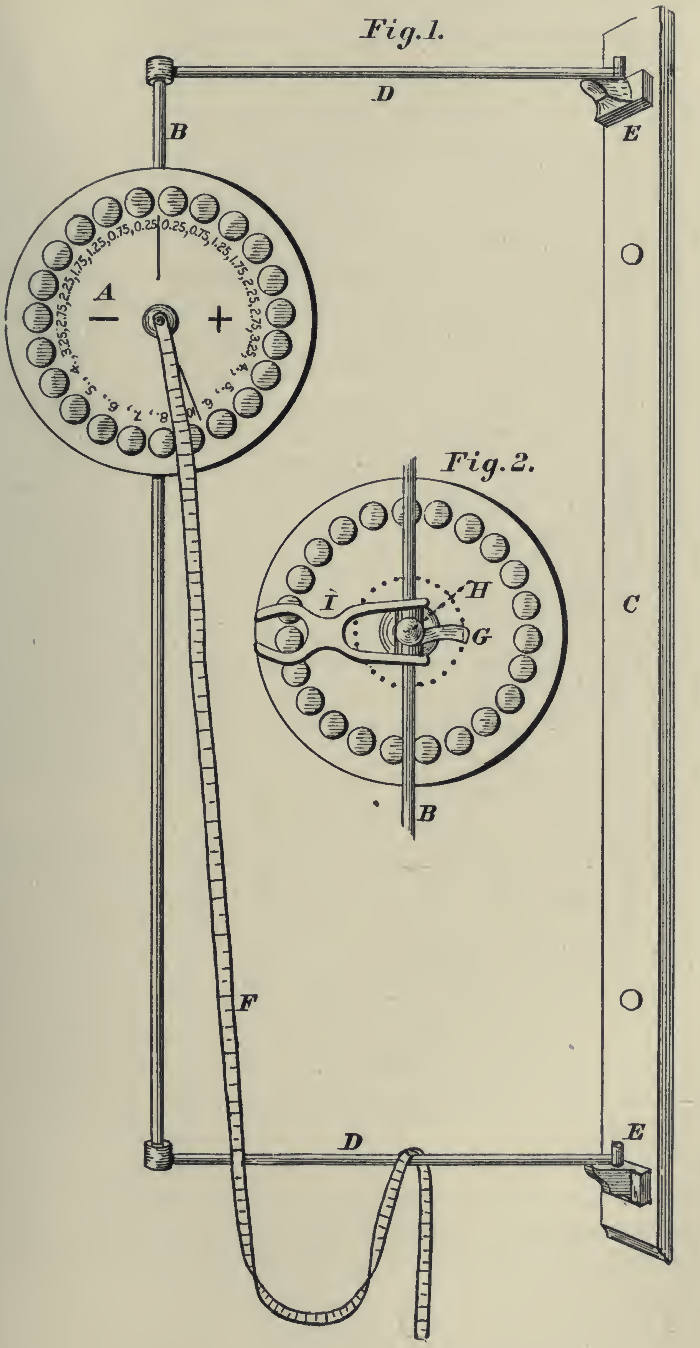

Fig. 1.

Fig. 2.

D
B
E
A
−
+
0.25, 0.75, 1.25, 1.75, 2.25, 2.75, 3.25, 4., 5., 6., 7., 8., 10
0.25, 0.75, 1.25, 1.75, 2.25, 2.75, 3.25, 4., 5., 6., 10
C
I
H
G
B
F
D
E

curacy of the method, a large number are no doubt debarred from its use by the lack of an easy method of employing it.

In using it in the manner most generally employed, it is necessary to fit a trial frame before the eye, and place lenses therein in succession until one is found which gives emmetropic movements to the shadow. This, however, is so consumptive of time and patience that the surgeon in the hurry of practice is likely to select some other and shorter method. It was for the purpose of facilitating this examination that I devised the simple apparatus described below, and which I exhibited first at the meeting of the American Ophthalmological Society, at Washington, in 1888. I have been using it since that time in my daily practice to my own complete satisfaction, and I have no hesitation in recommending it to the profession for the purposes intended. It consists of a hard-rubber disk, *A*, Fig. 1 (Fig. 2 being a back view), containing 23 lenses on its periphery—10 convex and 13 concave. This disk is movable on the brass-rod *B*, which is attached at either end to two other bass rods, *D D*, which turn on the pivots *E E* fastened to the wooden board *C*, which is attached to the wall of the ophthalmoscopic room. This enables the disk to be placed, by means of the screw *H*, Fig. 2, at any height, and to be turned and retained in any desired position, and the whole apparatus can be turned back against the wall when not in use.

The patient is placed in a proper position near the ophthalmoscopic lamp, and the disk is brought before the eye to be examined. By turning the disk on its axis all the lenses can be brought in rapid succession in front of the eye, and the one giving emmetropic movements to the shadow soon found. The revolving of the disk can be done by the patient or by the surgeon himself, who can easily reach it by a step from his examination distance of one to one and a half-metres. Of course the disk can be made large enough to contain any number of lenses. But those I have used are, according to my experience, ample for all ordinary cases, and keep the size of the disk to easily manageable dimensions. When other and stronger lenses are required, a lens from the trial-case of a number higher than the highest in the disk can be placed in the clip *I*, Fig. 2, which can be turned on the rod *B* to either side as desired, and the disk rotated as before, beginning at 0.25. The lens in the disk thus found is, of course, to be added to that in the clip, in order to have the full strength of the lens demanded. Not the least important use of the clip, however, is for holding the cylinder which has been selected for the correction of any existing astigmatism. This is very important, in many instances, for verifying the findings by other methods.

The graduated tape-measure *F* attached to the disk enables the observer to measure his exact distance from the eye under examination, which is necessary in the method as used by Jackson and others.

This apparatus, I claim, has the advantage over a somewhat similar disk devised by Doyne of Oxford, and exhibited at the International Medical Congress in Washington in 1887, in so far as it is less cumbersome, more easily manipulated, and is much less expensive. The disk can also be mounted on a stand, like a photographer's head-rest, which would allow of its being moved from place to place. Under certain circumstances this is the preferable form—for instance, where a special dark-room is not available. The instrument is made under both forms by Queen & Co., Philadelphia.

A NEW TREATMENT OF CHRONIC TRACHOMA.

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FOR NERVOUS DISEASES, LONDON.

(*With Drawings.*)

BEFORE proceeding to describe in detail the treatment I have adopted during the last two years with success in a large number of cases of chronic and subacute trachoma, I hope it will not be considered foreign to the subject if I endeavor to place before the reader the facts upon which I base my definition of trachoma and its allied diseases.

Were the forms of the disease clearly defined and understood by every one, such a course would be superfluous, but, unfortunately, this is far from being the case. I therefore may be pardoned if I try to state clearly the exact meaning to be attached to the words chronic trachoma, and preface the description of the operation by a few remarks on the nature of the disease, otherwise my method of treatment would be open to the objection of being credited with the cure of a disease which would have got well equally rapidly under any kind of treatment. Besides, I wish to be able to point out more clearly the class of cases in which modifications of the treatment may be desirable.

Trachoma naturally resolves itself into the acute and chronic forms, but as the former embraces or enters into a variety of closely allied diseases, such as oph. contagiosa, blennorrhœa acuta, and gonorrhœal ophthalmia; and as, moreover, the relationship between the trachomatous and the gonorrhœal poison would involve a discussion out of place in this paper, I shall omit all further reference to the acute form, especially as the treatment must be conducted on totally different principles.

Following the classification adopted by the German school, which is by far the simplest and most intelligible, I shall consider trachoma under the following heads:

First. A papillary or epithelial form corresponding to the chronic trachoma of Saemisch, a form in which an hypertrophy of the conjunctival papillæ is the prominent sign, but in which the follicles are hidden from view.

Secondly. A follicular or improperly termed "granular" form, in which the follicle forms the chief characteristic, corresponding to the conjunctivitis follicularis acuta of Saemisch, the blennorrhœa granulosa of v. Arlt, the trachoma folliculare of Horner, and the trachoma verum of others (Mandelstamm). Unfortunately, some authors call this latter the "papillary" form, which leads to great confusion and should only be applied to the first form, while others misname the follicles "granules," or "granulations," thereby confusing the discrete lymphoid follicles with the surface granulations. The first and second forms very often occur together and form the mixed form, "tr. mixtum" of Stellwag, in which the follicles lie in and beneath the hypertrophied or injected papillæ, though partly visible to the naked eye. For exaggerated and advanced conditions of this latter form, Stellwag has given the names trachoma diffusum and tr. sulzicum.

Lastly, we have to notice the disease known as follicular conjunctivitis—which many authors recognize as a variety of the second form above mentioned, but clinically it is to be distinguished as it is readily curable and never leads to those disastrous changes in the lids and corneæ so common in trachoma proper. In this follicular catarrh, the follicles are for the most part confined to the retrotarsal fold of the lower lid, although they undoubtedly occur in both lids. They are oval in shape rather than round, being smaller, more prominent, and more transparent than in trachoma proper, resembling vescicles rather than tubercles, and never found hidden in the deeper structures of the lid.

It has been repeatedly demonstrated that the characteristic feature of both these forms of trachoma is the follicle[1]; although in many cases of papillary trachoma the follicles are completely hidden to view by the swollen, gelatinous, or hypertrophied conjunctiva of the lids and retrotarsal folds, nevertheless they are always present, and may be readily found, if desired, by snipping a minute piece of the conjunctiva and subjacent tissue out of the lid, as I have proved for myself on several occasions. It is a simple and harmless test, and, if properly done, leaves no scar behind.[2]

Many different kinds of irritants will produce simple hypertrophy of the papillæ, and therefore one must never fall into the error of diagnosing trachoma by the mere presence of an inflamed lid covered with papillæ or "granulations," for every one must be familiar with cases where an atropine solution, or even ectropion of the lid exposed to the air, has produced a chronic catarrh with more or less velvety "granulations." In these cases it is only by observing the course of the disease and the actual demonstration of the follicle, that we can arrive at a true diagnosis. But although many mechanical irritants will produce these papillary hypertrophies, I am not aware that in the absence of micro-organisms they are sufficient to produce true trachoma. The few microscropic sections and cultivations that I have been able to make are in entire accordance with the now well-known observations of Rhein, that not merely several forms of micrococci, but several species of bacteria, are capable of inducing trachoma. I am inclined to the view that these pathological products, *the follicles*, are of the nature of a proliferating lymphoma, due to the irritation of the natural lymphoid tissue, probably by a microbe, and certainly by some prolonged source of irritation.

Whether the trachoma takes the form of a papillary hypertrophy, or a true follicular formation, depends largely on the resistance offered by the surrounding tissues, by the vitality of the lymphoid cells, and, lastly, by the nature and duration of the irritating cause.

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[1] Ohne Follikel kein Trachom! Aber aüch kein Follikular-Catarrh! Mandelstamm. *A. f. O.*, 1883, i., p. 76.

[2] I have never been able to satisfy myself that follicles ever occur as a natural product in healthy eyes which have not been subjected to prolonged irritation, although Dr. Rhein has demonstrated that in at least one other disease, viz., tubercle of the conjunctiva, follicles, identical in all respects with trachoma follicles, may be met with. *A. f. O.*, xxxiv., 3, p. 65.

We may, with Raehlmann,[1] conveniently divide the disease into three well-marked stages for the better explanation of the treatment.

The first stage is that of the formation and proliferation of the follicles, accompanied by more or less pannus.

The second stage is that of the fatty degeneration and secondary metamorphosis of the follicles, followed by ulcers and the formation of granulation tissue.

The final stage, that of scar formation, which is the natural termination of the disease, often leading to xerosis.

From a therapeutic point of view the first stage is by far the most important of the three, for it is the only one in which an absolute cure can be effected. It is a stage which may last for many years, varied by alternating periods of improvement and relapses.

Although the trachoma follicles are probably products of the irritation set up, still we have reason to believe they are at first rather a consequence or product than a cause of the disease, and it is only when they undergo degenerative processes, and so act as foreign bodies, that they set up an irritation and tend to slough out. As this will always occur sooner or later, our object in treating the disease must be to procure a speedy outlet for the follicles, and to effect such changes as will destroy the vitality of the organisms, or at any rate decompose or render harmless the poison present.

Now it struck me that the reason why the method of treatment hitherto adopted did so little towards relieving the patients was because the remedy applied was mainly a surface treatment, and whilst the astringent used, be it copper, zinc, silver, tannin, naphthol, or carbolic acid, benefited the surface and contracted the vessels, yet it failed to penetrate to the deeper structures of the lid, and especially beneath the fornix conjunctivæ, where the follicles abound. On the other hand, the heroic treatment with the galvano-cautery

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[1] See Volkmann's Sam. kl. Vorträge (1885). Ueber Trachom (No. 263), a most comprehensive article.

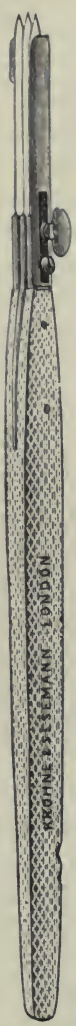

Fig 3

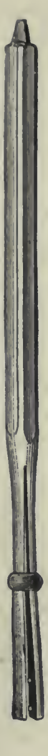

Fig 4

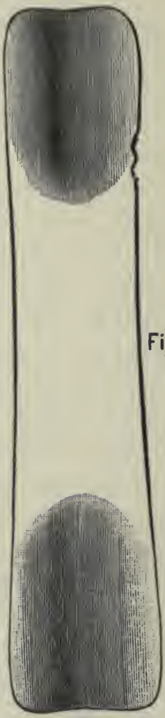

Fig 2

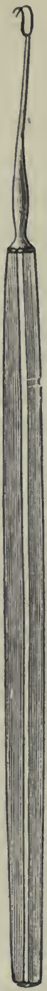

Fig 1

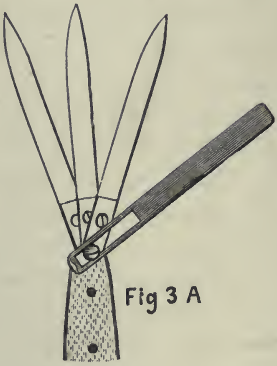

Fig 3 A

EXPLANATION OF FIGURES.

Fig. 1. Double bent hook, used for stretching the conjunctiva over the spatula.

Fig. 2. Spatula, curved so as to rest against the upper surface of the lid.

Fig. 3. A three-bladed sillonneur, with movable guard.

Fig. 3A. Showing the sillonneur open to enable the blades to be cleaned.

Fig. 4. Handle to hold the blades when required to be sharpened.

Fig. 5. The platinum electrolyser.

Fig. 5A. The platinum ends, showing how they may be moved aside for cleaning. Each blade works on a hinge (not seen in the wood-cut).

Fig. 6. Blunt button-shaped electrolyser.

The instruments have all been made to my design by Messrs. Krohne & Lesemann, Duke Street, Manchester Square, London.

led to such scarrings and contractions as to make the remedy as bad as the disease, and thus to entirely defeat the end in view.

In the course of practice it occurred to me that, by electrolysis, I could eliminate the follicles and reach their cause, and by employing needles as the terminals, I could penetrate the deeper layers. Experience taught me that more complete and radical methods would have to be adopted, and accordingly I had an instrument prepared wherewith to open up the deeper structures of the lid more thoroughly. From time to time I made various modifications both in the instruments themselves and in the method of using them, which it will be unnecessary to mention now. I will therefore proceed to explain the operation as I now practise it.

The Operation.

If the case be one of chronic trachoma of either the papillary or follicular variety, in the less advanced stages, before cicatricial changes have set in, I proceed as follows. The patient being placed under an anæsthetic—and he must be very completely narcotized, owing to the sensibility of the parts,—the upper lid is everted over the end of the vulcanite spatula (Fig. 2), and the conjunctiva kept tightly stretched over it by means of the double hook (Fig. 1).

This should be inserted close to the free edge of the mucous membrane of the eyelid. I have had the spatula made of vulcanite, it being essential that the same should be a non-conductor in order to protect the subjacent structures. Moreover, the two ends of the spatula are of different sizes to fit a large or small eyelid. These two instruments should now be firmly held between the finger and thumb of the left hand, while the three-bladed scalpel, or "sillonneur" (Fig. 3), is taken in the right hand. This should be held lightly like a pen, and the movable guard adjusted to a distance of 2 *mm*, 3 *mm* or 4 *mm* from the tip, according to the looseness of the conjunctiva, the œdema of the papillæ, and the general appearance of the lid—in other words, the more succulent the mucosa or the larger and thicker the papillæ, and the more swollen and congested the parts generally, the deeper must be the incision. On the other hand, the more the lid approaches the follicular variety, and the more anæmic the lid, the shallower the cuts need be. The incisions are best made parallel to the edge of the lid in a gentle curve, the first incision being made close to the edge and the others following in regular succession towards the retrotarsal fold. (See Fig. 7). About fifty small tufts of absorbent cotton-wool, previously dipped in an aqueous solution of boracic acid or hydronaphthol 1:1100, and then squeezed nearly dry, to be kept in readiness by the assistant, who should rapidly mop up the blood in the track of the sillonneur (3-bladed scalpel).

Having made a complete cut from corner to corner with the sillonneur, the next incision must be made in the track of the first; *i.e.*, the first blade of the sillonneur must run in the groove made by the third blade of the previous cut. The cuts must be made by a *finger* and not by *hand* motion, otherwise the cuts will not reach the further side of the lid; in other words, the instrument should be moved so as to become vertical towards the end of the incision. After sev-

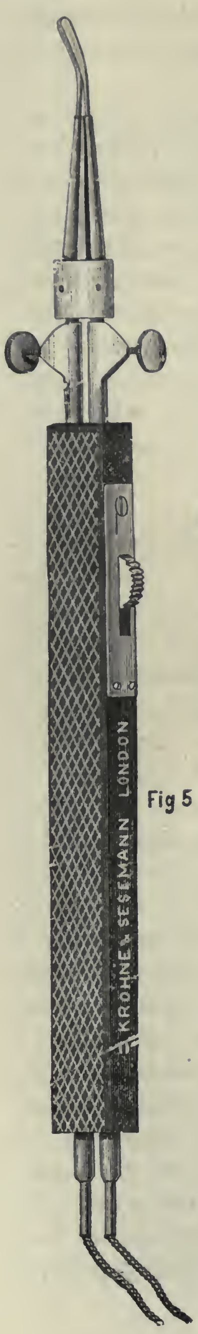

Fig 5

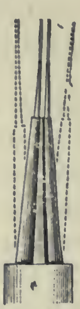

Fig 5a

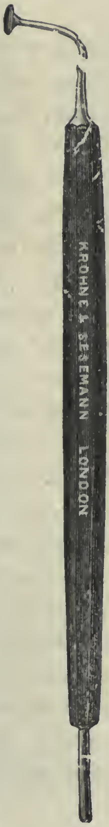

Fig 6

eral cuts have been made in this way, the hook must be released and reinserted farther away from the free edge, so as to put the swollen retrotarsal fold on the stretch. The cuts are to be continued several times in this manner, and the hook is to be reinserted at the posterior border of the cartilage, so as to get the whole of the retrotarsal fold over the end of the spatula; by this means the whole of the conjunctiva nearly up to the bulb can be incised. The bleeding is sometimes excessive, but can always be controlled by firm pressure with the cotton pads on the conjunctiva.

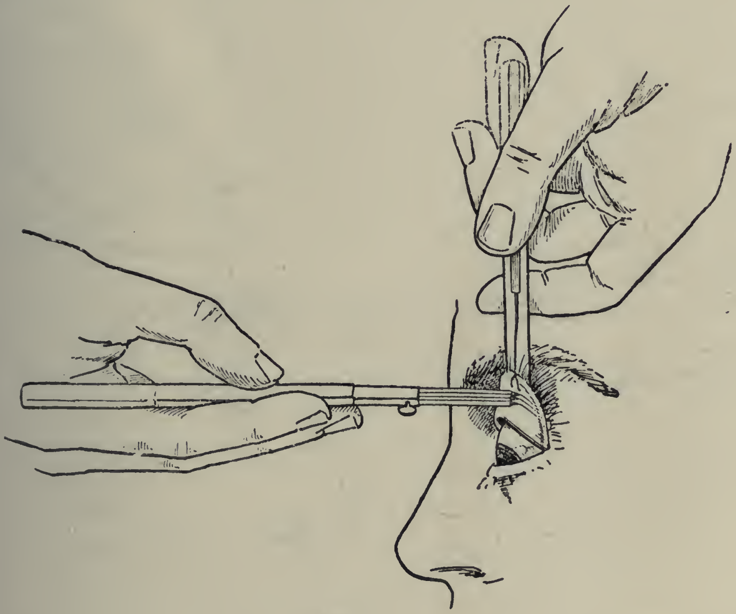

FIG. 7.

It is generally advisable to operate on both upper lids one day and both lower lids a few days later, as it is rare to find only one eye affected, and were both lids of one eye operated on the same day the inflammation set up might be excessive.

This part of the operation ought not to take more than three minutes for each lid.

We now come to the *second stage*, see Fig. 8.

The spatula being held under the everted lid as before, and the hook reinserted in its first position near the free margin, the electrolyser (Fig. 5 and 5 A) is now connected by wires with a battery. I have always used a Stöhrer's carbon and zinc battery of twenty cells. The instrument being held in the right hand, the lower edges of the two platinum blades are pressed firmly in the first two grooves made by the sillonneur and very slowly drawn along the furrows from end to end. About four or six cells (= about thirty milliampères) are used, and if in good working order the result will be at once seen by the thick foamy cream which arises round the blades. This is due to bubbles of hydrogen which are given off at the terminals (the platinum blades), and which, mixing with the blood and exudation, form the yellowish-looking froth. All the grooves are taken in turn, being slowly opened up along the lid from end to end.

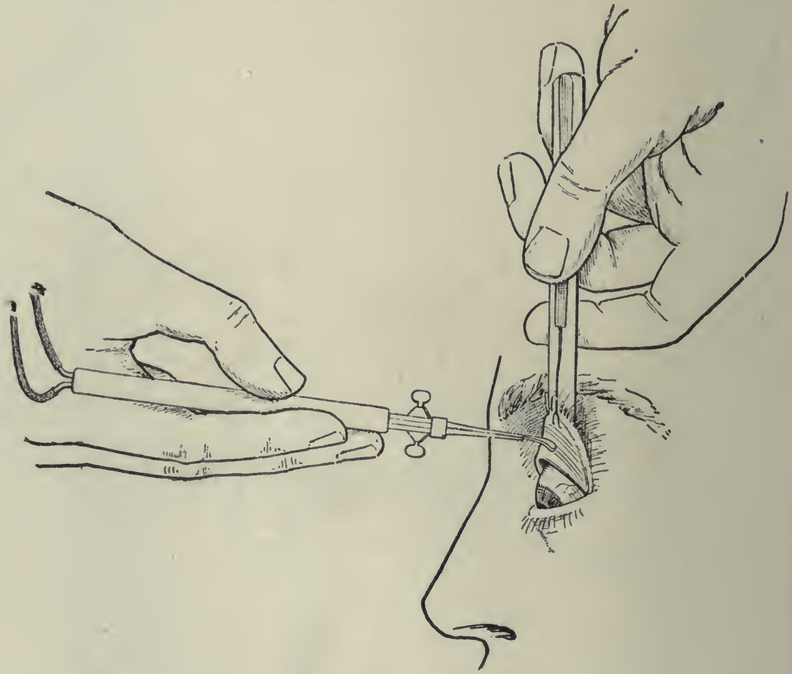

FIG. 8.

Frequently the spatula will have to be shifted to enable the lid to be put well on the stretch and to allow of the platinum blades reaching well into the corners. The foam all the time must be rapidly and repeatedly sponged away, and especially from around and between the blades. This stage of the operation takes about eight or nine minutes for each lid, as the blades have to be drawn through all the furrows very slowly from the free edge of the lid to the last incision made with the knife. It will be found that the electrolytic action tends to stop the bleeding rapidly, so no fear of after hemorrhage need be entertained.

I would remark here that I have used currents of very varying strengths, and find that strong currents should be avoided. If many cells are used, *i.e.*, more than six or eight of the Stöhrer battery, the conjunctiva becomes slightly carbonized, giving to the lid an ashy-brown appearance. This not only sets up an unnecessary amount of inflammation, but, acting more like a cautery, greatly retards healing, causing slight sloughs, and often leads to small ulcers of the cornea as well. I have found as the result of innumerable trials that from four to six cells give the most satisfactory results.

Should the retrotarsal fold be very loose and much thickened, it may at this juncture be found advisable to dissect out a narrow strip of conjunctiva along the whole length of the fold. This may be hooked up by means of Galezowski's double forceps, or more simply, and I think quite as well, by the ordinary fixation forceps. I found that the conjunctiva was apt to drop between the two sets of jaws in Galezowski's pincers, and to avoid this I had a pair made with an intermediate set of jaws one millimetre shorter, *i.e.*, nearer the shank than the side jaws, but I ultimately relinquished them for the simpler process of pinching up the fold with a pair of ordinary fixation forceps and cutting away the conjunctiva beneath the jaws with a small pair of scissors narrow and curved on the flat. Unless the conjunctiva be very loose, it is best not to remove the strip of conjunctiva, or at best only to remove a very narrow one—1 *mm* or 1½ *mm*,—else too much of the fold may be picked up, and the movements of the eyeball, if not limited, at any rate slightly disfigured by an ugly fold when the eyes are rolled upwards. This of course can only happen in the case of the lower lid, as the upper one overlaps too much to render any fold visible without everting the lid. But in the majority of cases the incisions made with the sillonneur will be found to do all that is necessary both for opening up the hiding-places of the follicles which crowd the recesses of the fold, and for cutting off the supply of blood from the vessels which help to produce the pannus. I have rarely found it necessary to cut this strip away from the lower fold, and I now only use it exceptionally in the case of the upper one.

Last Stage of the Operation.

Both lids are now washed free from blood, sprinkled with a five-per-cent. cocaine solution and dusted with calomel. Finally they are smeared over with an ointment made of vaseline and hydronaphthol, 1 to 800, and the eyes bandaged up with thick, moistened compresses. Should the inflammation during the next twenty-four hours be at all severe, which may be judged by the swelling of the eyelids, I order iced compresses to be applied and frequently changed.

I have during the last few months been using an ointment containing one part of hydronaphthol (β naphthol to 800 of vaseline, a treatment which I find is much recommended by Professor Panas in cases of chronic trachoma. I only use the proportions 1:400 to 1:800 deeming them sufficient for all purposes, but Professor Panas uses it up to one per cent., but then he uses it on the lids without previous operative treatment.[1]

For forty-eight hours the discharge from the lids is often considerable and many of the follicles slough out, but between the second and third day a decided change for the better sets in and the wounds rapidly heal. Usually at the end of a week the lids have so far healed that nothing remains to be seen except the nearly healed wounds mostly hidden by a slight velvety injected surface. If the current has been too strong or too long applied, the sloughing may continue for at least a week, and thick cicatricial bands will be ultimately found, irregular in direction, and rarely following the course of the incisions. Although as yet I have never had a case in which these artificially made scars produced any entropium, yet they always cause a slight unevenness on the lid; it is prejudicial to the rapid clearing up of the pannus. But if the strength of the current be attended to, and a very good idea can be formed by the amount of foam and the change of color in the conjunctiva, these scars will never happen. Formerly I was accustomed to prolong the action of the current until the lid gradually changed to a brownish color, but subsequent cases have taught me the bad effect of allowing the current to play over any part too long. It is a good plan to insert a galvanometer in the track of the wires, if there is any doubt about the strength of the current, and Stöhrer provides an excellent and very portable one for this purpose, but a little experience will enable the operator to dispense with it, as it is rather in the way, and one has not much time to look at it. The current should not exceed fifty milliampères.

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[1] *Archives d'Oph.*, January–February, 1889.

Formerly I found it necessary to repeat the operation on both upper and lower lids but by using a weaker current, and at the same time using the platinum blades more thoroughly and systematically, I have found one operation quite sufficient.

As soon as the wounds have healed up I wash the lids with a hot boracic lotion each day, afterwards smearing them over with vaseline or hydronaphthol ointment, 1:800. This should be continued for at least three weeks. It is not necessary to keep the patient in bed during the treatment, as I have operated on a large number of out-patients and they have apparently done quite as well as those whom I have taken into the wards, provided they had good diet and well ventilated rooms. Great care should be taken not to touch the cornea during the operation, as rather severe ulcers and abscesses may ensue by neglecting this. But with ordinary care there is no danger whatever. At the same time, should any sign of corneal ulceration be seen, the operation must be postponed, for, as every one knows, corneal ulceration and abscess are among the frequent complications of trachoma.

An important adjunct to the treatment is attention to the bodily health of the patient. Trachoma in this country at least seems to flourish chiefly among ill-nourished and strumous subjects. As regards internal medicines, those which may be looked upon as supplementary to foodstuffs, like cod-liver oil and the phosphates and hypophosphite syrups of Parrish and Fellowes, are the most satisfactory. Children rarely refuse the oil when administered with equal quantities of the above syrups after meals. About a fortnight or three weeks after the operation it is advisable to send the patient to the seaside or the hills, to prevent the possibility of re-infection (unfortunately a common occurrence). The highest spot above the sea level is the best. Chibret says that at an altitude of 200 metres trachoma loses its contagiousness, and the follicles do not increase. How far this is true I do not know, but the testimony appears unanimous as to the close inverse relationship between altitude and the frequency of trachoma. It is a well-known fact that as we follow the rivers from their mouths to their sources trachoma diminishes, in proof of which we need only mention the familiar examples of the Rhine, the Loire, and the Nile, and (as I have been personally informed), in a still more pronounced degree, among the natives of the Congo and the Niger.

Cases of trachoma folliculare must be treated somewhat differently from the papillary variety. In the former, a very weak current of two to four cells only should be used, nor should the application be prolonged or the incisions so deep. I have in my notes records of two cases in which a great deal of inflammation followed, together with ulceration of the cornea from ignorance of this fact. In one of the cases, which, fortunately, is the only case of the kind I have met with, the patient had an ulcer of the cornea at the time of the operation, which spread so rapidly afterwards as to become uncontrollable, and in spite of every care ended in total leucoma. The other eye, which was operated on at the same time and in the same manner, recovered perfectly, all trachoma disappearing and the pannus cleared up sufficiently to enable the patient, from counting fingers, to read J 4 and $\frac{20}{50}$ Sn a month afterwards. In the other case—a private one—an ulcer of the cornea followed, which caused a great deal of trouble for some time afterwards.

The first of these two cases was among my earliest, and greatly discouraged me at the time. I have performed the operation ninety-six times since, and omitting the two cases previously mentioned, have never known any evil consequences follow.

I have never yet had a case of trachoma, mixtum or papillare, of which this treatment has failed to answer my best expectations. I suppose the reason why the uncomplicated follicular type does so badly under severe treatment is owing to the absence of the pronounced vascularity which is so characteristic of the other forms. It might be thought that partial gangrene or at least extensive ulceration might follow such repeated incisions, but this practically never occurs, and it can only be accounted for by the very abundant vascular supply and extensive network of anastomoses with which the lids are furnished.

I have been repeatedly asked whether entropion or ectropion does not ultimately follow the operation. I can answer positively that I have never yet seen a case in which the slightest abnormal curvature has followed in consequence of the operation.

The second stage (ulceration of the follicles) is best met by frequently dusting the lids with calomel powder and anointing with vaseline and hydronaphthol, after previous irrigation with hot (100° F.) two-per-cent. boric-acid solution. This should be done every day.

In cases where the palpebral fissure has been much reduced, by the adhesion of the lids at the outer canthus, after repeated attempts to maintain the fissure permanently open to its natural width by various plastic operations, I have at length succeeded in obtaining a partial but permanent separation of the united parts of the lid by the following operation, which is merely an adaptation of Baker's operation for separating webbed fingers. I procure a piece of silver wire, the size of a No. 2 nasal probe, and file one end until it is double-edged like a suture-needle. I then pass the pointed end of the wire between the united lids and the globe, and with the finger of the other hand overlapping the outer rim of the orbit as a guide, push the wire through the seam of the lids and skin. I then pull half the wire through, cut off as much as necessary with cutting-pliers, and with two blunt wire pliers, attached to each end, bend the wire so as to form a ring the size of a sixpence. I then pass a narrow piece of strapping, two and a half inches long, through the ring, with the adhesive sides facing each other. These being pressed together over the join of the ring, and the free end being placed on the cheek, the strapping is kept in its place by extra strapping laid over it. By this means movement of the ring is possible without the cut ends being able to slip round and fret the conjunctiva. After being worn for a month, so as to form a permanent cicatrix round the wire, I divide the tissues with a sharp knife up to the ring, which now drops out, and with some fine silk sutures draw the conjunctiva over the wound of each lid, to prevent reunion. I always make the fissure longer than normal, as at least half is sure to become obliterated in course of time. The lids should be pulled away as far apart as possible every day for weeks.

In the stage of xerosis I have obtained considerable benefit by washing out the conjunctival sac and lids daily with a hot two-per-cent solution of boracic acid, and scraping away with a knife all the white froth from the globe. This froth I have repeatedly submitted to the microscope, and it appears to consist almost entirely of epithelial cells, cocci, and short rods, as figured and described by Leber (*A. f. O.*, 1883, iii., p. 225, and Tab. 8). This removed, I drop on to the eye a solution of boracic acid in glycerine to keep it moist. Sometimes the patients prefer to have the eye frequently powdered with calomel.[1]

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[1] The calomel powder which is blown in (preferably by a ball atomizer) should be very pure. I always have mine prepared by stirring it in a large volume of distilled water for some time, leaving it to settle—decanting and drying gently over a water-bath. The calomel when quite dry is finally triturated in a mortar. This treatment eliminates all the perchloride which may be present. As Dr. Weeks has shown (KNAPP'S ARCHIVES, vol. xvi., p. 377), calomel is a powerful antiseptic, although this property is not due to its partial conversion into $HgCl_2$ by the action of the salts in the tears, as I have shaken up calomel with the lachrymal secretion for hours without finding a trace of perchloride afterwards.

The superficial and interstitial opacity which exists is best treated by the direct application of a platinum button (Fig. 6) to the cornea, the instrument being connected to the negative pole of the Stöhrer's battery opened to about four cells, the positive pole at the same time placed anywhere on the forehead. Each application should last about four minutes, and is generally quite painless if cocaine be used first, as the nebulous cornea is comparatively insensible.

I think it is unnecessary to burden this paper with an account of the numerous cases which I have treated. Suffice it to say they embrace every stage of trachoma, while a considerable number have previously been the round of the hospitals and dispensaries, many having been under treatment for four, five, or even ten years previously without receiving permanent benefit. I am greatly indebted to the medical superintendents of several of our London workhouses and infirmaries for keeping me supplied with cases, and especially to Dr. Milne, who placed all his trachoma patients in Her Majesty's Hospital, Stepney, under my care.

It would be ungrateful of me to conclude this paper without expressing my warmest thanks to Mr. Lawson, who first permitted me to demonstrate the operation at Moorfields, and to Professor Tweedy for his valuable aid and advice. I should also like to thank Dr. Sartain, of Philadelphia, who, during his stay in England, kindly assisted me in many of the operations.

REPORT ON A THIRD SERIES OF ONE HUNDRED SUCCESSIVE CATARACT EXTRACTIONS WITHOUT IRIDECTOMY.[1]

BY H. KNAPP.

THE series of cataract extraction to be reported in the following pages, were all performed by myself in the N. Y. Ophthalmic and Aural Institute, between Jan. 5, '89, and Jan. 24, '90. During my absence in March and August, a number of extractions were made at the "Institute" by Dr. R. O. Born. They are not included in the following report; nor are there included eleven operations of my own which do not fall within the limits of our subject. I shall speak of these eleven operations separately, so that no case that has been operated on by me within this period, shall be passed over in silence. The eleven cases set apart were as follows:

TWO CASES in which the functional examination revealed intraocular lesions, probably detachment of the retina, were admitted to operation with a doubtful prognosis. Their recovery was without essential disturbance; in the one a small prolapse of the iris occurred, but did not prevent a smooth healing process. The patients, though preserving the normal shape of their eyes, did not recover sight. I have often operated under similar circumstances, and have had the pleasure of obtaining useful sight in a certain number of patients. In some cases, I think, it is not possible to diagnosticate detachment of the retina before the operation. Perception of light may be preserved in the whole field of vision if the retina is raised only in small folds. When there is no cataract we make the diagnosis in such cases not so much from the examination of the visual field as with the ophthalmoscope.

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[1] The First Series was published in vol. xvii. (1888), p. 51, etc., the Second Series in vol. xviii. (1889), p. 1, of these ARCHIVES. The different series are the immediate continuation of one another, so that no case operated on is omitted.

A THIRD case was the extraction of a chalky lens dislocated into the vitreous and complicated with total detachment of the retina. The eye was blind. The operation was undertaken only for cosmetic reasons and to remove the irritation of the whole eye produced by the movable lens. The operation, which completely answered our expectations, is described in the previous number of this volume, p. 53.[1] I should not omit to mention a case of extraction of a secondary cataract in which the lens had shrunk after two needlings, years ago. The patient, Mr. Wright, thirty-one years old, addicted to drink, having his face full of acne pustules, was unruly during the operation, and made a sudden movement with his head while I was drawing the lens out with a Mathieu forceps. The cataract came out clean, but with escape of vitreous. The pupil was clear and central, but the eye was lost by suppuration of the vitreous, evidently through infection from his diseased skin and conjunctiva, in spite of careful sterilization of both with soap and bichloride of mercury. Dense secondary cataracts are, perhaps, the most dangerous objects to deal with, division and incisions being mostly impracticable, always dangerous. Extraction seems to be relatively the safest procedure. It may be total, if the resistances are not great, or partial, *i.e.*, a portion is drawn out of the wound and cut off.

There were seven cases in which I made iridectomy during the operation. Briefly detailed they are as follows:

CASE 1.—Pat. Dunn, æt. 67, operated in his bed, regular section. Large lens expelled without difficulty. Several attempts to obtain a round pupil were unsuccessful. Tendency to prolapse. Iridectomy made. Recovery undisturbed. S $\frac{20}{100}$. Discission. S $\frac{20}{70}$.

CASE 2.—A. M. Stähle, æt. 68. Conditions as in previous case. Recovery. S $\frac{20}{70}$.

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[1] On the Extraction of Lenses Dislocated into the Vitreous.

CASE 3.—Mrs. Rath, æt. 63. Splitting of capsule with knife during the section ; a *peripheric part of iris cut off*, the central part drawn out with forceps and cut ; during the cleansing of the eye, which was made very difficult by nervousness of patient and forcible closure of the lids, some *vitreous escaped.* Recovery smooth. S $\frac{20}{100}$. Disc. S $\frac{20}{40}$.

CASE 4.—Mrs. Long, æt. 62. A peripheric portion of iris fell before the knife during a sudden pressure of the patient and was exsected. The central portion was then likewise removed. Extraction of lens as usual. *Purulent kerato-iritis.* Eye lost. As a possible cause of the infection it may be mentioned that, in order to disinfect a somewhat congested conjunctiva, a strong solution of nitrate of silver had been brushed in without my knowledge.

CASE 5.—Noah Selbig, æt. 91. Cataract complicated. On account of great narrowness of pupil, an iridectomy was made. Expulsion regular, followed by unusual collapse of the cornea. *Purulent kerato-iritis* without any assignable cause. Loss of eye.

CASE 6.—Mrs. Roberts, æt. 56. Patient utterly unruly, screaming like a child, and pressing lids together ; therefore iridectomy. Expulsion of lens without accident. Recovery with good sight.

CASE 7.—Mrs. E. Rhoden, æt. 57. Cataract swollen by imbibition, removed without accident, but the iris lay in the wound and, the patient being uncontrollable, was abscissed. Cleansing of pupil. No reaction. S $\frac{20}{200}$. Disc. S $\frac{20}{50}$.

Intercurrent with the first 100 cases of simple extraction there were 17 in which I made iridectomy; in the second 100 cases there were 13 ; in the third series only 7. Instead of finding an iridectomy desirable in a greater number of cases, as some operators have done during the last years,[1] I have constantly reduced the number. There will always be special indications for iridectomy, but I do not think that I shall ever cease to practise simple extraction as a rule.

With regard to the

Mode of Operating,

the experience of the last year has not been without influence on certain points in the technique.

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[1] In the Report of the Eye Clinic of Prof. H. Sattler, of Prague, for the year 1889, I read, p. 4: "Extractions according to von Graefe, 213 ; extractions with a small flap without iridectomy, 4."

1. **Copious injections into the anterior chamber, with any liquid whatever, seemed to irritate.** I have therefore limited the injection to letting a small quantity of a very mild antiseptic—a solution of one half per cent. of chloride of sodium in boiled water would probably be the best—flow through the whole extent of the section. I introduce the point of a fine syringe into the anterior chamber, and, pressing gently on the piston, let the water flow over the wound from within outward. When the liquid is forced through the wound by directing the stream from outside in, particles that may be in the conjunctival sac or in the wound will be driven into the anterior chamber, as the entrance of bubbles and particles of blood-clots frequently demonstrate. I have spoken of this before, but like to repeat it, as the reverse method is followed by men whose opinions I consider very valuable.

2. **Liberal irrigations with corrosive sublimate,** $\frac{1}{5,000}$, produced indelible staining of the cornea in two cases, making the operation in one case (No. 33) result in failure, in the other (No. 37) in the recovery of moderate vision only. I do not think that we should condemn these irrigations altogether, but I should make them rigorously only where a septic condition of the conjunctiva—as in the unconquerable chronic dacryocystitis—seems to be present; whereas, under aseptic conditions, they may be employed mildly, and the intraocular injections totally omitted. Sure it is that the most painless and smoothest recoveries are obtained when neither irrigation nor injections are made. But then we must be sure that the region of the operation, in and outside the lids, and every thing that comes in contact with the wound, is perfectly aseptic. The cleansing of the lids, especially the ciliary margins, with soap and bichloride seems to me very essential. In regular operations this may not matter much, but if there be escape of vitreous there is a good nutrient fluid spread from the lids uninterruptedly into the eye. I have never seen this better illustrated than in the case of Mr. Wright, the extraction of the shrunken lens, mentioned above (p. 281).

Any thing that produces a congestion, be it exposure to dust, lack of sleep, irritants such as corrosive sublimate or nitrate of silver in a strength to cause hyperemia, lymphatic exudation, and exfoliation of epithelium, *i.e.*, food for bacteria, should be strictly kept off the eye for days previous to the operation. I like to have my patients one or several days in the hospital, in good air, perfectly resting their eyes and bodies, doing nothing except washing their faces and eyes with soap two or three times daily. If, by this treatment, a congested conjunctiva has become pale and shining, I consider the patient in a fit condition for the operation.

3. **I do not adhere exclusively to one particular kind of dressing.** Nobody will contest the truth of the old dogma that one of the chief requisites for securing primary union in any wound is rest of the part, as absolute as we can have it without interference with the patient's general health. If I were to be operated on for cataract, I would sacrifice a great deal of my comfort for the smallest gain in security. In winter, and with quiet patients, the binoculus, as far as I can judge, fulfils the conditions of rest better than any other dressing. With nervous patients, and in summer with almost all, I place a patch of corrosive-sublimate gauze on the eye, a small pad of absorbent cotton on it, and hold them in position by strips of plaster. Strips of isinglass plaster immediately on the lids are, to some patients, on account of their stiffness, more unpleasant than either of the two modes of dressing mentioned above. Apart from this, the removal of the plaster by tearing it off requires somewhat forcible pulling of the lids, which, in a recently operated eye, may disturb the wound, and if we soak the plaster first it consumes an inordinate time. Should secretion occur, the plaster is incontestably inferior to the gauze. During the hot weather of this summer, I have closed a greater number of eyes with isinglass plaster, simply, as recommended by C. E. MICHEL of St. Louis,[1] J. J. CHISOLM of Baltimore, and others. The results, thus far, have been favorable.

I think also that it is safer to let the patient lie in bed the first days after the operation than to give him the freedom of the house. When one eye is not bandaged, the patient will look about, and the closed eye will accompany the movements of the other.

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[1] See his paper: "Dark Rooms and Bandages discarded in the after-treatment of Cataract Operations, Iridectomies, etc.," in these ARCHIVES, Vol. XV., p. 318, etc., 1886.

4. As is well known, **hypostatic pneumonia** will occasionally develop in decrepit people after extraction of cataract. Until lately I have had no such case in my whole practice—I have made over two thousand cataract operations—and last year two cases, fatal, as almost all are, occurred within a short period. The one was a man of forty-three years of age, the other a woman of seventy-four. In neither were there any promonitory symptoms. The disease declared itself over night, as the usual pneumonia does, from which, however, it is distinguished by the purely bloody character of the sputum and the absence of bacteria. In neither of our cases were bacteria found in the sputum. In both the pneumonia set in when the eyes were cured and the patients out of bed—in the man, on the tenth day after the operation on the second eye; in the woman, on the twelfth day. Both had made excellent and undisturbed recoveries from the operations. What rules can be deduced from these cases? Certainly to let old people not lie too long in the same position, and to sustain their forces. In neither of our cases had these rules been disregarded, for both were well, had no reaction from the eye operation, enjoyed good appetite, and no food was forbidden to them. Besides, they both were up when the pneumonia set in. I think that Snellen goes too far in recommending old people—as I am informed he does—to get up one or two days after the operation and sit in the yard. When a patient complains of the bed, let him sit up in his room, which can always be ventilated sufficiently to let him breathe fresh air. In institutions where many patients are crowded in the same room, as is the case in most hospitals, this, of course, is different.

5. With regard to the **technique of the operation, I shall change the section in so far as to make it less peripheric.** I do not hesitate to place myself on record in asserting that **one of the greatest errors** *ever committed in the history of cataract extraction was the introduction of the peri-*

pheric section by J. Jacobson, late professor of ophthalmology in the University of Königsberg. I had enough of the peripheric section with all its irido-cyclitic consequences when I practised extraction with iridectomy. Since 1886 I have followed De Wecker and Panas in making the section exactly in the transparent margin of the cornea, rather with a leaning toward a conjunctival flap. My experience leads me to infer that the old dogma, enunciated by Mackenzie and others, is true, *i.e.*, the farther away from the centre you make an opening into the cornea, the more you will have incarceration and prolapse of the iris. There are two oculists who claim scarcely ever to see hernia of the iris after cataract extraction. I mean Galezowski of Paris and C. S. Bull of New York. Galezowski says that he does not open the eye before a week elapses; Dr. Bull lays stress on operating on his patients in their beds. I have done the one and the other, and have had hernias; but another feature which Galezowski and Bull have in common, is that both place the apex of the section within the cornea, Galezowski 2*mm*, as he stated five years ago, and Dr. Bull, when I questioned him recently, said that the apex of his section lay always in the clear cornea. I have shifted occasionally toward the old peripheric section with a conjunctival flap, and whenever I did so prolapses were more frequent. A conjunctival flap has its advantages. The wound closes more quickly, and is much better protected from secondary infection. Sections lying wholly in the cornea frequently show a "riding flap"; that is, the distal flap projects over the proximal. This produces a groove. Slow union of the wound and tardy restoration of the anterior chamber are the consequence. The latter is unimportant, but the former, creating a nidus for bacteria, is dangerous. My sections of late have been less peripheric than before, and I intend giving this, the Galezowski section, a fair trial. The liability of prolapse of iris is the weak point in simple extraction. Dr. Bull says he has never had real prolapse, *i.e.*, hernia, but he confesses a considerable number of incarcerations. Besides, in thirteen of the one hundred cases which he has reported there was escape of vitreous, a number which is far greater than the average. These points can be settled only by extensive observation, and as no man can do every thing, we should all work in concert and learn from one another.

6. Regarding the **management of the capsule** my experience may be of value. In 64 successive cases I have opened the capsule with the knife in its passage through the anterior chamber, the old procedure of Wenzel (1808), recently revived by Galezowski, Gayet, and Mooren. I have abandoned this procedure as a general method, and have published my results from it, its advantages and disadvantages, in the last volume of these ARCHIVES, 1889, p. 169. I shall therefore not refer to it now. Since then I have practised the **peripheric opening** of the capsule, by introducing a delicate, sharp cystitome under the upper part of the iris and splitting the capsule as I used to do in the extraction with iridectomy. When I formerly tried this procedure I thought that the exit of the lens was less easy, but now, on extensive trial, I find that the lens escapes quite readily, and that I have no longer adhesions in different parts of the pupil, but either a single adhesion in the upper part or none at all. Thus I gain a clean area in which to practise the final clearing of the pupil, the secondary discission of the capsule. The cystitome needles which Tiemann and Meyrowitz made for me are delicate and sharp, so as to cut and not tear, similar to the cystitomes I used for the peripheric capsulotomy in Graefe's extraction, with the only difference that the stem of the new cystitome is not straight, but slightly bent, because it has to be introduced into the anterior chamber and then turned. The manœuvre is not difficult.

7. Another point to which I have given much thought is the **extraction of a piece of the anterior capsule,** a procedure which seems to be in very extensive use with the most renowned operators in Europe. In hundreds of cases I removed a central piece of the anterior capsule when I practised Graefe's method. The results were not favorable; shreds of capsule were but too apt to lodge in the section and be the passage-ways of infection, ushering in plastic and purulent inflammations. With the simple extraction this is not to be feared, because the iris is interposed between

capsule and corneal section. But I have found other difficulties in this procedure. I have not always succeeded in what I attempted to do, or, in case I succeeded, have torn the capsule too extensively, especially toward the corneal section, causing vitreous to follow the escape of the lens. In general I have had more satisfaction in opening the capsule peripherally, expelling the lens and then extracting the thickened portion of capsule, than in attempting to extract it before the expulsion of the lens. I know only a few of the European models of capsule forceps, among which that of Schweigger. Its teeth spread out well, but they project when the branches are closed, and the instrument is thick and clumsy. Mathieu's forceps are neat, but their teeth being parallel to the branches, they easily fail to grasp a flat and yielding membrane. I have therefore sought to modify these forceps in such a way that they can be introduced and withdrawn without showing projecting parts, but on opening in the eye the branches will revolve on their longitudinal axes making the teeth project downward and on gentle pressure grasp the capsule, return to a horizontal position when closed, and carry the grasped portion out without grating on the iris or the lips of the corneal wound. The principle is this: When the branches are closed, the teeth, being horizontal, do not project, as will be seen in the drawing, (Fig. 1, *a*), where the branches are parallel.

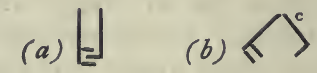

FIG. 1.

In *b*, where the branches open on the joint *c*, the teeth project downward. This does not serve our purpose, but it will do so when we attach to the end-points of the branches (*b*) prolongations at right angles, and put the teeth on the inner side of these prolongations. Then the teeth will be perfectly covered when the branches are closed, and will project downward when opened. The instrument under consideration may not possess any new features, but I have tried it, and find that it answers the purpose. The present model which Geo. Tiemann & Co. have spared no pains in making as simple and efficient as possible, is shown in the accompanying figure (Fig. 2).

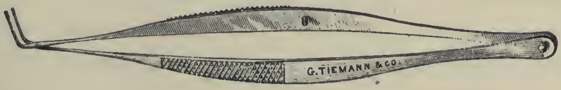

FIG. 2.

Among the **peculiarities and complications of cataract,** we may mention:

1. Two cases (27, 31) of *nuclear sclerosis*, in which the cortex was transparent. The whole lens came out without difficulty, requiring no manipulation to clear the pupil, and the visual results were good ($\frac{20}{30}$ in each).

2. Four cases (6, 29, 68, 80) had old *maculæ of the cornea* which had no influence on the operation, but decreased the vision ($\frac{20}{50}$, $\frac{20}{100}$, $\frac{20}{200}$, $\frac{20}{100}$).

3. *Atrophy of the optic disc*, two cases (2, 53), with S $\frac{20}{200}$ and $\frac{10}{200}$, the poor result greatly owing to irregular astigmatism from prolapse of iris. I may mention that slight degrees of optic-nerve atrophy, with poor sight, for instance $\frac{20}{200}$ to $\frac{20}{50}$, in good optical conditions, has not been noted, but is not at all infrequent in cases of cataract extraction. The pallor of the optic disc is the only thing to account for the amblyopia.

4. *Atrophy of choroid*, one case (5), very marked with S $\frac{15}{200}$. A number of other cases with more or less extensive crescents and opacities of the vitreous have not been specially noted.

No.	Name, Age, Health.	Cataract ; Eye.	Operation, Healing Process, Duration of Treatment.	Primary S.	Secondary Operation after Extraction, Duration of Treatment.	Ultimate S.	Remarks.
1	Mrs. Grebe. 41. Good.	R. (right eye). Soft,	**1889.** Jan. 5th. Regular. 12 days.	$\frac{20}{50}$	Discission in 6 weeks. 5 days.	$\frac{20}{20}$	Before disc. the capsule was evenly spread through the pupil ; fundus veiled ; metamorphopsia.
2	Geo. Mausert. 55. Potator.	Hard, ripe, complicated.	Jan. 9th. *Severe iritis.* Traumatic *prolapse of iris* on 4th day, cut off a few hours after its occurrence. Pupillary membrane.	$\frac{1}{200}$	Disc. in 6 months. Healing smooth. Pupil clear. 4 days.	$\frac{20}{200}$	Later $\frac{6}{200}$; white atrophy of opt. disc;
3	B. Soltau. 32. Good.	Hypermature, good.	Jan. 9th. Thickened capsule, incised and removed. Expulsion laborious. No reaction whatever. 15 days.	$\frac{20}{40}$			
4	P. H. Ward. 43. Somewhat anæmic.	Ripe.	Jan. 11th. Regular. No reaction. On 7th day had a chill, followed by pneumonia of left lung, later of right, died on 17th day of *hypostatic pneumonia*, the diagnosis, made *intra vitam* by the bloody expectoration and absence of bacteria, verified by the autopsy.	$\frac{20}{40}$			The other eye had been operated on 2 weeks previously with excellent result. Patient had lost one leg during the war.
5	Mary Sullivan. 55. Good.	L. Hard ; complicated, excessive myopia with atrophic choroiditis.	Jan. 26th. Regular. No reaction. 14 days.	$\frac{15}{200}$			
6	Mary Glass. 42. Good.	Morgagnian, complicated with mac. corn.	Jan. 28th. Regular. 14 days.	$\frac{20}{70}$	Disc. 18th day. Mild iridokeratitis. 16 days.	$\frac{20}{50}$	
7	Ch. Ott. 58. Good.	Ripe, good.	Jan. 30th. Breech delivery of lens. No reaction. 13 days.	$\frac{20}{30}$	Disc. 23d day. 5 days.	$\frac{20}{20}$	
8	Mrs. Reynolds. 52. Good.	Ripe, good.	Feb. 2d. 4th day pupil round and central. No reaction. Hurt eye on 6th day. *Pupil oblong*, iris not protruding. 18 days.	$\frac{20}{100}$	Disc. 2 months. 5 days. $\frac{20}{70}$.	$\frac{20}{30}$	
9	Chas. Renard. 72. Mentally unsteady.	Ripe, good.	Feb. 6th. Regular. 14 days.	$\frac{20}{200}$			Pupil clouded. Patient did not return for a discission.
10	Sarah Young. 73. Good.	Not quite ripe, good.	Feb. 9th. Reg. 14 days.	$\frac{20}{100}$	Disc. 21st day. 5 days.	$\frac{20}{50}$	On 6th day.
11	Mrs. E. Brown. 60. Good.	Ripe, good.	Feb. 9th. Reg. 14 days.	$\frac{20}{100}$	Disc. 9 months. 3 days.	$\frac{20}{50}$	Defective choroidal pigmentation. Optical conditions perfect.

12	E. C. Bull. 81. Feeble.	Nuclear sclerosis, good.	Feb. 13th. Reg. Pupil slightly oval. 30 days.	$\frac{20}{50}$			
13	J. Wally. 61 (negro). Good.	Ripe, good.	Feb. 14th. Reg. 13 days.	$\frac{20}{100}$	Disc. 6 weeks. 5 days.	$\frac{20}{50}$	On 6th day.
14	Mrs. Stinson. 66. Good.	Ripe, good.	Feb. 15th. Reg. 13 days.	$\frac{20}{100}$	Disc. 6 weeks. 5 days.	$\frac{20}{20}$	
15	Mary Sullivan. 55. Good.	R. Ripe, complicated, large crescent.	Feb. 16th. Reg. 21 days.	$\frac{20}{200}$		$\frac{20}{40}$	In 8 months.
16	Mary Oswald. 60. Heart disease.	Ripe, good.	Feb. 27th. Reg. 11 days.	$\frac{20}{50}$			
17	Sarah Sanders. 63. Good.	Ripe, good.	March 29th. Operated in her bed. Some difficulty to get the pupil round. 4th day iris under conjunctival flap; coughed much in the morning. No reaction. 11th day, sudden pains at night, for an hour; ant. chamb. empty (injury). Slow recovery, with moderate *prolapse of iris.* 6 weeks.	$\frac{18}{200}$ Eight weeks $\frac{20}{100}$	Disc. 4 months. 6 days.	$\frac{20}{70}$	Wound cystoid. No particular irritation. Seen 11 months after extr., the prolapse almost disappeared.
18	Aug. Halbrecht. 38. Good.	Ripe, good.	March 30th. Op. in bed; reg. 14 days.	$\frac{20}{70}$		$\frac{20}{20}$	Upper part of pupil clear, lower slightly veiled.
19	Mrs. E. Lowe. 63. Good (negress).	L. Ripe, good.	April 3d. Op. in bed; reg. 17 days.	$\frac{20}{70}$	Disc. 4 weeks. 4 days.	$\frac{20}{20}$	
20	Therese Buschendorf. 53. Good.	R. Ripe, good.	April 4th. Op. in bed; reg. 21 days.	$\frac{20}{70}$	Disc. 5 weeks. 5 days.	$\frac{20}{40}$	In 2 weeks, some flakes in pupil.
21	C. Gallenger. 70. Good.	L. Ripe, good.	April 8th. Op. in bed; reg. 12 days.	$\frac{20}{100}$	Disc. 5 weeks. 5 days.	$\frac{20}{40}$	
22	Auguste Rosa. 35. Good.	Ripe, good.	April 10th. Op. in bed; reg. 14 days.	$\frac{20}{100}$	Disc. 4 weeks. 5 days.	$\frac{20}{100}$ $\frac{20}{40}$	In 5 days later.
23	Arth. Spiegel. 21. Good.	Soft, ripe.	April 10th. Op. in bed; reg. 12 days.	$\frac{20}{50}$	Disc. 4 weeks. 5 days.	$\frac{20}{40}$	In 5 days.
24	Jas. Conkling. 59. Good.	Ripe, good.	April 12th. Op. in bed; reg. 13 days.	$\frac{20}{100}$	Disc. 4 weeks. 5 days. On 8th day an attack of *glaucoma*, cured with eserine.	$\frac{20}{40}$	
25	Miss Rockwood. 80. Good.	Ripe, good.	April 20th. Op. in bed. Patient refractory. Difficulty in getting the lens out. *Mild iritis.* Pupil round and central, a few filiform adhesions. 28 days.	$\frac{20}{200}$ later $\frac{20}{70}$	Disc. 2 months. 4 days.	$\frac{20}{30}$	

No.	Name, Age, Health.	Cataract; Eye.	Operation, Healing Process, Duration of Treatment.	Primary S.	Secondary Operation after Extraction, Duration of Treatment.	Ultimate S.	Remarks.
26	Thos. Trembath. 58. Good.	R. Ripe, good.	April 24th. Op. in bed; reg. 15 days.	$\frac{20}{40}$		$\frac{20}{20}$	Capsule spread evenly through pupil, a break in centre where knife had passed. Fundus clear.
27	C. McCann. 42. Good.	L. Centre opaque, cortex clear, good.	April 24th. Op. in bed; reg. Lens came out entire. 15 days.	$\frac{20}{50}$	Disc. 7 weeks. 4 days.	$\frac{20}{30}$	
28	Thos. McCarty. 62. Good.	Ripe, good.	April 26th. Reg. 14 days.	$\frac{20}{50}$	Disc. 4 weeks. 4 days.	$\frac{20}{30}$	
29	Miss Cath. Howe. 68. Good.	Ripe, complicated corneal opacities from trachoma.	May 3d. Op. difficult on account of contraction of lids from *trachoma*. Some discharge. Healing of wound undisturbed. 15 days.	$\frac{13}{200}$		$\frac{20}{100}$	Pupil clear and round.
30	Mrs. Gert. Millet. 52 (Costa Rica). Good.	R. Ripe, good.	May 6th. Op. reg. 4 weeks.	$\frac{20}{200}$	Disc. 6 weeks. No reaction. 4 days.	$\frac{20}{100}$ later $\frac{20}{50}$	
31	Mrs. Mart. Lyons. 60. Good.	Nuclear sclerosis, good.	May 8th. Reg. 16 days.	$\frac{20}{200}$	Disc. 4 weeks. 4 days.	$\frac{20}{30}$	
32	Mrs. McNealy. 55. Good.	Ripe, good.	May 11th. Reg. 14 days.	$\frac{20}{50}$	Disc. 5 weeks.	$\frac{20}{20}$	
33	Adolf Wrede. 68. Good.	Ripe, good.	May 11th. Reg. Wound irrigated freely with corros. subl. 1:5000. 4th day, *iris in wound*, cornea diffusely milky, whitish opacity, like a cloud before pupil in ant. chamber. On 13th day protruding iris cut. No inflammation. Cornea and pupil opaque.	Fingers at 3 ft.		$\frac{1}{1000}$	Seen again a year later. Cornea whitish-opaque. Shape of globe and T good. (Corr. subl. reaction.)
34	Wm. Dunn. 57. Good.	Ripe. Good.	May 9th. Reg. Slow restoration of ant. ch. (7 days); 14 days.	$\frac{20}{50}$			
35	John Schmolch. 56. Good.	Hypermature.	May 13th. Descemetitis from free washing out. Ant. ch. Cornea and pupil cleared up. 22 days.	$\frac{20}{200}$	Disc. 4 months. 4 days.	$\frac{20}{30}$	
36	Charlotte Bick. 61. Good.	Nuclear sclerosis.	May 15th. Reg. 15 days.	$\frac{20}{30}$			
37	Mrs. P. Cohn. 70. Feeble.	Ripe. Good.	May 18th. Ether. Op. regular. *Mila iritis.* Cornea hazy and pupil dull. Patient restless and cranky. 11 days.	$\frac{3}{200}$		$\frac{6}{200}$	A month later when last seen, cornea still hazy. Too much sublimate irrigation.
38	Joseph Samech. 52. Good.	Ripe. Good.	May 18th. Reg. 13 days.	$\frac{20}{50}$	Disc. 24 days. 3 days.	$\frac{20}{20}$	

39	Mrs. Hager. 77. Good.	R. Ripe. Almost Good.	May 18th. Op. laborious on account of nervousness of patient. Reg. 14 days.	20/50	Disc. 6 weeks. 4 days.	20/20	
40	Mrs. Kerr. 58. Good.	Ripe. Good.	May 20th. Reg. 15 days.	20/100	Disc. 20 days. 4 days.	20/30	In 5 days.
41	Mrs. R. Mulligan. 74. Good.	Ripe. Good.	May 20th. Op. reg. 4th day, small *prolapse*, traumatic; blood in chamber. Prol. cut 9th day. 18 days.	10/200	Disc. 6 weeks. 3 days.	20/50	
42	Mrs. French. 74. Feeble.	Ripe. Good.	May 22d. Healing of eye regular. On 11th day hypostatic *pneumonia*. *Diea* 14 days later. 25 days.	20/50			
43	Mrs. Hunt. 78. Feeble.	Ripe. Good.	May 25th. Reg. 14 days.	20/50	Disc. 6 weeks. 4 days.	20/30	
44	Dan. Moylan. 43. Good.	Cat. lactea. Iris atrophic.	May 27th. Extr. *within capsule* by external manipulation. Pupil round and central. Healing regular. 14 days.	20/100		20/70	Pupil clear. Floating opacities in vitreous.
45	Mr. Mack. 68. Good.	Ripe. Good.	May 28th. Tendency of iris to prolapse on the slightest pressure. 4th day, patient injured eye, hyphæma, pupil central. 14 days.	15/200	Disc. 6 weeks. 4 days.	20/40	Vitreous slightly hazy.
46	Mrs. Fitzgerald. 62. Good.	Ripe. Good.	May 29th. Reg. 12 days.	20/30			Pupil central, unusually clear.
47	Jean Klumbach. 62. Good.	Not quite mature. Good.	May 30th. Reg. 19 days.	20/40	Disc. 6 weeks. 5 days.	20/30	
48	Sam. Birnbaum. 21. Good.	Soft. Good.	June 1st. Reg. 17 days.	20/30			
49	Mrs. Smith. 78.	Ripe. Good.	June 5th. Reg. *Summer dressing of eyes* (a patch of sublimate gauze, covered with some absorbent cotton, held in position by 2 strips of court plaster). 14 days.	20/200	Disc. 5 weeks. 4 days. Round, regular, black pupil.	20/50	5 months later attacks of *glaucoma*, repeating as in the prodromic stage of ordinary inflammatory glaucoma, relieved by eserine, but ultimately in March, 1890, the ant. ch. being shallow and the iris convex, $+T_1$, a regular upward iridectomy was made, which resulted in permanent recovery.
50	Mr. Southwick. 69. Good.	Hypermature. Good.	June 10th. Reg. 10 days.	20/70	Disc. 4 weeks. 5 days.	20/20	
51	Ann Carrey. 48. Good.	Ripe. Good.	June 12th. *Peripheric piece of iris exsected, lens passed* out *through this opening.* Recovery smooth. 13 days.	20/40	Disc. 6 weeks. 4 days.	20/20	

No.	Name, Age, Health.	Cataract; Eye.	Operation, Healing Process, Duration of Treatment.	Primary S.	Secondary, Operation after Extraction, Duration of Treatment.	Ultimate S.	Remarks.
52	S. Austin. 66. Good.	Ripe. Good.	June 12th. Reg. 12 days.	$\frac{20}{50}$	Disc. 24 days. 5 days.	$\frac{20}{20}$	Had S $\frac{15}{15}$ after 2d operation. Resumed work too soon and too persistently. Had irido-cyclitic irritation 4 weeks. Recovery with good sight. $\frac{20}{20}$. June 4th, 1890.
53	F. Miller. 69. Good.	Ripe. Complicated.	June 17th. Traumatic *prolapse of iris.* 15 days.	$\frac{5}{200}$		$\frac{10}{200}$	Atrophy of od. The prolapse has remained free from irritation, but is large. The upper part of the cornea is pushed forward so that near the centre the cornea is horizontally wrinkled, resembling a saddle surface. Regular and irregular astigmatism marked.
54	Taylor, Dr. 62. Good.	Ripe. Good.	June 18th. Reg. 11 days.	$\frac{20}{200}$	Disc. 5 weeks. 4 days.	$\frac{20}{20}$	
55	Mrs. G. Kelly. 86. Good.	Ripe. Good.	July 1st. Opening of capsule with cystotome insufficient. Expulsion of lens laborious. Some *escape of vitreous.* Recovery smooth. 12 days.	$\frac{20}{70}$		$\frac{20}{50}$	
56	Mrs. G. Millett. 52. Good.	L. Ripe. Good.	July 1st. Some *iris, falling before knife,* exsected. 21 days.	$\frac{20}{70}$	Disc. 7 weeks. 3 days.	$\frac{20}{40}$	
57	Eliza Farwell. 48. Good.	Immature. Good.	July 2d. Reg. 14 days.	$\frac{20}{70}$	Disc. 3 weeks. 5 days.	$\frac{20}{20}$	
58	Mrs. Schepp. 78. Good.	Ripe Good.	July 2d. Some adhesious. 16 days.	$\frac{20}{70}$	Disc. 6 weeks. 5 days.	$\frac{20}{30}$	
59	Jos. Beermender. 46. Good.	Tremulous. Opac. cornea. Atrophy iris.	July 3d. Capsule drawn out with Mathieu's forceps. 3d day *small prolapse.* Healing good. 15 days.	$\frac{15}{200}$		$\frac{20}{50}$	Prolapse cut in 6 weeks.
60	Mrs. Dubréus (Haiti). 45. Good.	Ripe. Good.	July 3d. Peripheric section. Small piece of iris cut. *Glaucoma* developed on 6th day; shallowness of ant. chamb. Eserine and pilocarpine reduced tension, but wound gradually bulging.	$\frac{5}{200}$		$\frac{20}{200}$	Seven weeks after operation wound slightly elevated. Ant. chamber almost empty. Pupil tolerably clear. Od. just seen, not excavated, Tn, Fn. Later news by letter indicates that eye remains subject to relapses of irritation.
61	Mich. Enscoe. 58. Good.	Ripe. Good.	August 19th. Reg. 14 days.	$\frac{20}{70}$	Disc. 6 weeks. 5 days.	$\frac{20}{40}$	

62	Sarah Use. 57. Good.	Ripe. Good.	August 20th. On 7th day she knocked her head against a wall. *Hyphema.* No prolapse. Blood gradually absorbed. Pupil round and central. 27 days.	$\frac{20}{70}$			
63	Jer. Egan. 57.	Ripe. Good.	August 23d. Reg. 12 days.	$\frac{20}{50}$		$\frac{20}{20}$	In 3 weeks.
64	Louis Loos. 37. Good.	Ripe. Good.	September 3d. Reg. Capsule divided by a horizontal incision, by passing cystitome behind the upper part of the iris.	$\frac{20}{50}$		$\frac{20}{20}$	In 4 weeks. Capsule thin and even. Horizontal, superior cystotomy employed as a rule in the following cases.
65	Mrs. P. Ward. 57. Good.	Ripe. Corneal opacity.	September 3d. Reg. 15 days.	$\frac{20}{100}$	Disc. 18 days. 4 days.	$\frac{20}{40}$	Pupil and background clear.
66	Phil. Hardt. 49. Good.	Dislocated. Fair.	Sept. 18th. Extra. with capsule. Some *prolapse of vitreous.* No irritation. Recovery good. 21 days.	$\frac{20}{70}$			
67	Mrs. Sullan. 60. Good.	Ripe. Good.	Sept. 20th. Reg. 22 days.	$\frac{20}{200}$		$\frac{20}{40}$	In 2 months. Pupil and background perfectly clear.
68	Mary Bach. 69. Good.	Ripe, complicated, corn. opacities, and old trachoma.	Sept. 24th. Reg. 17 days.	$\frac{5}{200}$	Disc. 2 months. 5 days.	$\frac{20}{200}$	
69	Chr. Rettberg. 72. Good.	Ripe. Good.	Sept. 27th. Reg. 12 days.	$\frac{20}{70}$		$\frac{20}{20}$	In a month.
70	W. Lawrence. 55. Good.	Ripe. Thickened Capsule.	Oct. 3d. Reg. 14 days.	$\frac{3}{200}$	Disc. 4 weeks. 4 days.	$\frac{20}{40}$	
71	Ann Jones. 51. Good.	Ripe. Good.	Oct. 5th. Reg. 14 days.	$\frac{20}{100}$	Disk. 4 weeks. 4 days.	$\frac{20}{40}$	
72	Mary Colloran. 70. Good.	Ripe. Good.	Oct. 12th. Reg. 13 days.	$\frac{20}{70}$		$\frac{20}{50}$	
73	Mrs. McQuade. 60. Good.	Ripe. Good.	Oct. 14th. Reg. 14 days.	$\frac{20}{200}$			
74	Mr. N. Rose. 80. Good.	Ripe. Good.	Oct. 16th. Reg. 15 days.	$\frac{20}{20}$			
75	Ch. Schöttel. 70. Good.	Ripe. Good.	Oct. 16th. Reg. 14 days.	$\frac{5}{200}$			
76	Mrs. Wise. 55. Good.	Ripe. Good.	Oct. 18th. Reg. On 12th day fell down stairs. Slight bruises. Eye uninjured. 22 days.	$\frac{20}{50}$			
77	Cath. Travers. 71. Good.	Ripe. Good.	Oct. 23d. Reg. 17 days.	$\frac{20}{70}$	Disc. 16 days. 9 days.	$\frac{20}{50}$	

No.	Name, Age, Health.	Cataract; Eye.	Operation, Healing Process, Duration of Treatment.	Primary S.	Secondary Operation after Extraction, Duration of Treatment.	Ultimate S.	Remarks,
78	Mrs. Car. Valoroshi. 65.	Swollen by imbibition. Good.	Nov. 1st. Small prolapse of iris. No reaction. 15 days.	$\frac{20}{100}$			
79	Dr. McCaw. 66. Good.	Ripe. Good.	Nov. 2d. Reg. 15 days.	$\frac{20}{50}$	Disc. 20 days. 4 days.	$\frac{20}{20}$	
80	Mrs. Rowland. 70. Good.	Ripe, dacryocystit. chronic.	Nov. 4th. Op. healing of wound regular; the tear-sac carefully pressed out; the secretion, which by previous treatment had been reduced to a minimum, taken up with cotton, and junctival sac is washed out twice daily with corr. subl. 1 : 5,000. 20 days.	$\frac{20}{100}$			
81	Ther. Miller. 78. Good.	Ripe. Good.	Nov. 9th. Reg. 14 days.	$\frac{20}{70}$	Disc. 4 weeks. 3 days. And again 3 weeks later. 4 days.	$\frac{20}{70}$	Vitreous still cloudy. Pupil clear.
82	Jas. Mahone. 60. Good.	Hypermature. Good.	Nov. 11th. Reg. 21 days.	$\frac{20}{50}$	Disc. 5 weeks.	$\frac{20}{40}$	
83	Mrs. Coholan. 47.	Thickened capsule.	Nov. 12th. Extr. in capsule tried. Capsule broke. Lens expelled. Capsule drawn out with forceps. *Escape of a drop of vitreous.* Recovery without irritation. *Incarceration of iris.* 14 days.	$\frac{20}{40}$		$\frac{20}{50}$	4 weeks.
84	Jos. Heartley. 74. Good.	Ripe. Good.	Nov. 13th. Reg. 13 days.	$\frac{20}{70}$			
85	Mary Cleary. 60. Good.	Ripe. Good.	Nov. 15th. Reg. 15 days.	$\frac{20}{100}$	Disc. 4 weeks. 4 days.	$\frac{20}{70}$	In 4 days.
86	Leon. Bauer. 44. Good.	Ripe. Good.	Nov. 18th. Normal pupil until 5 days, when he hurt his eye with his finger. Small prolapse. No irritation. 18 days.	$\frac{20}{70}$		$\frac{20}{50}$	
87	Daisy Green. 19. Good.	Soft immature.	Nov. 20th. Disc. discharged 7th day. In 6 days returned with the ant. chamber filled with lens masses. $+T_1$. Regular extraction at once. Relief. Discharged in 10 days, with a round pupil and a good eye.	$\frac{20}{30}$		$\frac{20}{20}$	

88	E. Miller. 69. Good.	Ripe. Good.	Nov. 23d. Reg. 18 days.	$\frac{20}{50}$		$\frac{20}{40}$	
89	Mrs. Hoggson. 54. Good.	Ripe. Good.	Nov. 30th. Reg. 15 days.	$\frac{20}{70}$	Disc. 4 weeks. 4 days.	$\frac{20}{20}$	4 months later still $\frac{20}{20}$. Pupil and background clear as crystal. (Ideal result.)
90	Mary Donnelly. 55. Good.	Ripe. Perception of l. poor, especially in upper part of F.	Dec. 9th. Reg. 14 days.	$\frac{20}{200}$			Apparently better V. Patient's mind weak.
91	Carol. Kahn. 74. Good.	Ripe. Good.	Dec. 11th. Reg. 16 days.	$\frac{20}{200}$			
92	Mich. Schnidler. 67. Good.	Hypermat. Good.	Dec. 11th. Reg. 14 days.	$\frac{20}{200}$	Disc. 5 weeks. 4 days.	$\frac{20}{70}$	In four days.
93	Mrs. Mary Detz. 65. Good.	Ripe. Good.	Dec. 11th. Reg. 12 days.	$\frac{20}{100}$			
94	Mrs. A. Davis. 50. Good.	Hypermat. Good.	**1890.** Jan. 4th. Part of capsule removed with Schweigger's forceps. Moderate iritis. 17 days.	$\frac{20}{100}$	Disc. 5 weeks. A band remains in centre of pupil. 8 days.	$\frac{20}{50}$	
95	Mrs. Sullivan. 67. Good.	Morgagnian. Good.	Jan. 8th. Reg. 16 days.	$\frac{20}{200}$	Disc. 5 weeks. 4 days.	$\frac{20}{50}$ $\frac{20}{20}$	In 4 days. In 3 months.
96	Mrs. Wolfsburg. 64. Good.	Ripe. Good.	Jan. 17th. Severe pain 1st night. *Central prolapse.* 19 days.	$\frac{20}{40}$		$\frac{20}{40}$	June 1st. Prolapse free from irritation, gradually contracting. S. good.
97	S. Moffat. 69. Moderate.	Ripe. Good.	Jan. 17th. Reg. 18 days.	$\frac{20}{40}$			
98	John Smith. 62. Good.	Ripe. Good.	Jan. 20th. Reg. 14 days.	$\frac{20}{50}$			
99	Ferd. Baumer. 67. Good.	Ripe, thick, irregular capsule. Good.	Jan. 20th. Reg. 14 days.	$\frac{20}{200}$	Disc. 33 days. 11 days.	$\frac{20}{30}$	In 3 months. Large circular staphyloma post.
100	Jas. S. Smith. 53. Good.	Ripe. Good.	Jan. 24th. Pat. unruly, coughing, disturbing bandage. Pupil oval. No incarceration. 22 days.	$\frac{20}{200}$			

Anomalies during the Operation.

1) "*Breech delivery*," *i.e.*, lower part of lens turning up and escaping first, posterior surface turned toward the cornea, one case (7) result good.

2) *Extraction within capsule*, two cases (44), S $\frac{20}{70}$, floating opacities in vitreous; and (66) escape of vitreous. Recovery of good sight, $\frac{20}{70}$.

3) *Exit of lens through peripheric exsection of iris*, one case (51). Recovery smooth. S $\frac{20}{20}$.

4) *Anterior capsule drawn out*, two cases (59, 94), in the former, on third day, small prolapse of iris. Of late I have oftener extracted pieces of the anterior capsule and have seen it followed by prolapse of vitreous, probably because I tore the suspensory ligament of the lens.

5) *Escape of vitreous* occurred three times, namely, besides the above mentioned case 66, in case 55, after a laborious expulsion of the cataract through an insufficient section; recovery smooth, S $\frac{20}{50}$; and in case 83, in which extraction within capsule was tried, capsule broke; capsule drawn out with forceps after expulsion of lens; escape of a drop of vitreous; incarceration of iris. S $\frac{20}{50}$.

6) *Iris fell before the knife and was cut* in two cases, in case 51, as mentioned above, at 3), and in case 56. Recovery and sight good in both.

Anomalies of the Healing Process.

1) Cases of iritis have been rare since I discarded the central opening of the capsule, substituting for it the opening along the upper border of the lens (see above, p. 287). In a case of *severe iritis* (2) with traumatic prolapse of iris a pupillary membrane formed (S $\frac{1}{200}$). It was discinded in 6 months with S $\frac{20}{200}$. *Mild iritis*, 3 cases (25, 37, 94) are mentioned, doing no damage to vision. There was a goodly number with one or two small posterior adhesions, without inflammatory irritation worth mentioning. They had no influence on the clearing of the pupil either spontaneously, or by discission.

2) There were several cases of *secondary hemorrhage* into the anterior chamber, all traumatic, all without consequence.

3) *Permanent haziness of cornea*, from too much corrosive sublimate irrigation, two cases, reported above, p. 283.

4) *Death from pneumonia* in two cases, Nos. 4 and 42 (see p. 285).

5) *Glaucoma*, 3 cases. In one (60) it followed the extraction on the sixth day, and led to, or rather was a symptom of, protracted irritation. The other two occurred after subsequent cystotomy, and are spoken of below (p. 301).

6) *Prolapse and incarceration of the iris*, the *bête noire* of simple extraction, occurred in eight cases. They were as follows:

(2) Traumatic on 4th day, cut off a few hours later. Severe iritis. $S = \frac{1}{200}$. Disc. $S = \frac{20}{00}$.

(17) Traumatic, on 11th day. Cystoid wound. No irritation. $\frac{20}{70}$.

(33) Small, not protruding, seen on 4th day, cornea and ant. chamber hazy from too much corr. subl. irrigation (see above, p. 283.). $S \frac{1}{1000}$.

(41) Traumatic, small, 4th day, cut on 9th. $\frac{20}{50}$.

(53) Central, moderate, cornea saddle-shaped, no irritation, $S \frac{10}{200}$, optic-nerve atrophy.

(59) Small, 3d day, cut in 6 weeks. $S \frac{20}{50}$.

(83) Incarceration. No reaction, $S \frac{20}{50}$, without secondary operation.

(96) First night. No reaction, gradually contracting. $S \frac{20}{40}$.

This result is not so bad. There was no eye lost, and only one, No. 53, had a badly misshapen cornea, which might be improved by cutting the prolapse, but the result would be futile on account of the presence of optic-nerve atrophy.

There was no suppuration in this series of extractions without iridectomy, but there were three cases of it among the intercurrent operations, as was stated above. The one was the extraction of a secondary (shrunken) cataract, the two others were operations with iridectomy. I do not mean to introduce this as an argument against the combined method, but the experience gained from the present series of cases invalidates again the opinion, still held by many oculists, that the iridectomy diminishes the number of severe inflammations, especially suppurations. My experience leads me to believe that just the opposite is true.

Visual Results.

	Primary V.	Ultimate V.
$\frac{20}{20}$	in 1 case	in 21 cases
$\frac{20}{30}$	“ 5 cases	“ 15 “
$\frac{20}{40}$	“ 8 “	“ 21 “
$\frac{20}{50}$	“ 20 “	“ 18 “
$\frac{20}{70}$	“ 18 “	“ 8 “
$\frac{20}{100}$	“ 17 “	“ 5 “
$\frac{20}{200}$	“ 16 “	“ 8 “
$\frac{18}{200}$	“ 1 case	
$\frac{15}{200}$	“ 2 cases	“ 1 case
$\frac{13}{200}$	“ 1 case	
$\frac{10}{200}$	“ 1 “	
$\frac{6}{200}$	“ 1 “	“ 1 “
$\frac{5}{200}$	“ 4 cases	“ 1 “
$\frac{3}{200}$	“ 4 “	
$\frac{1}{200}$	“ 1 case	$\frac{1}{1000}$
	100 cases	100 cases

The column of ultimate vision represents the results of the latest examinations, be they after or without a subsequent capsulotomy. I should mention that in 25 cases primary results only had been entered. Full visual acuteness was reached in 23 %, about as many as in the first series, less than in the second. Such variations may occur. They probably depend more on the greater or smaller number of complications than on differences in the operative procedure and the nursing, though I am far from underrating the effect of the latter. The difference is also owing to the smaller number of subsequent divisions of the capsule made in the third series, namely, 55, against 74 in the second series. Another difference worthy of mention is the fact that in the second series there was no case of full vision without a subsequent division of the capsule. In the third there were six cases, one even as a primary result; in the five others a lesser primary acuteness rose to the full standard spontaneously.

Subsequent Cystotomy.

A secondary operation was made in 53 cases, a smaller number than before, but I am sure that many patients of this series will present themselves later to have their sight improved by a secondary operation. This operation consisted in each case in the splitting of the capsule with a knife-needle according to the method which I have followed for years and repeatedly described. None of these operations had a bad effect. In a few cases it led to inflammatory irritation (cyclo-hyalitis), which, however, yielded in each case, without impairment of vision. Glaucoma followed the discission in case 24 on the 8th day, but was cured by eserine instillations. In case 49 glaucoma developed five months after the discission. Not yielding to eserine a regular iridectomy upward was made. Viscid (healthy) vitreous escaped after the incision, proving that vitreous was in the anterior chamber. The patient was permanently cured.

The following table will show in what **degrees of visual acuteness discission was resorted to, and what were the visual results obtained:**

S
$\frac{20}{30}$, 1 case converted into $\frac{20}{20}$.
$\frac{20}{40}$, 2 cases " " $\frac{20}{30}$, $\frac{20}{20}$ respectively.
$\frac{20}{50}$, 11 " " " $\frac{20}{20}$ 6 ×, $\frac{20}{30}$ 2 ×, $\frac{20}{40}$ 2 × $\frac{20}{50}$ 1 ×.
$\frac{20}{70}$, 11 " " " $\frac{20}{20}$ 4 ×, $\frac{20}{30}$ 1 ×, $\frac{20}{40}$ 3 ×, $\frac{20}{50}$ 2 ×, $\frac{20}{70}$ 1 ×.
$\frac{20}{100}$, 13 " " " $\frac{20}{20}$ 1 ×, $\frac{20}{30}$ 2 ×, $\frac{20}{40}$ 4 ×, $\frac{20}{50}$ 4 ×, $\frac{20}{70}$ 1 ×, $\frac{20}{100}$ 1 ×.
$\frac{20}{200}$, 9 " " " $\frac{20}{20}$ 2 ×, $\frac{20}{30}$ 3 ×, $\frac{20}{50}$ 2, × $\frac{20}{70}$ 1 ×, $\frac{20}{100}$ 1 ×
$\frac{18}{200}$, 1 case " " $\frac{20}{70}$.
$\frac{15}{200}$, 1 " " " $\frac{20}{40}$.
$\frac{10}{200}$, 1 " " " $\frac{20}{50}$.
$\frac{5}{200}$, 1 " " " $\frac{20}{200}$.
$\frac{3}{200}$, 1 " " " $\frac{20}{40}$.
$\frac{1}{200}$, 1 " " " $\frac{20}{200}$.

In all cases but one (where $\frac{20}{70}$ remained $\frac{20}{70}$) the operation increased the vision.

If we add the visual acuteness in the 53 cases before the discission and after it, we arrive at the aggregate gain of visual acuteness by the primary operation (extraction), the secondary operation (discission), and by both combined.

Primary vision of the 53 cases sums up to 12.95 full visions, which, at an average, gives each patient $\frac{1}{4}$ of the normal standard, *i.e.*, very near $\frac{20}{70}$.

After the secondary splitting of the capsule the aggregate visual acuteness of the 53 cases sums up to 32.53, *i.e.*, almost $\frac{20}{30}$ at an average. This result is really very gratifying, and may be expressed as follows:

The average visual result of extraction is $\frac{20}{70}$, which by subsequent cystotomy is converted into $\frac{20}{30}$.

This expresses, of course, only the experience of one operator. It remains to be seen how far it has general validity. Deducting the average primary result, $\frac{20}{70}$, from that obtained by the discission, $\frac{20}{30}$, we see that extraction gives the patient V $\frac{20}{70}$, and discission adds to it $\frac{20}{50}$—that is, more than he obtained by the extraction. The same result, computed in a similar way, was arrived at in my second series of 100 extractions.[1]

If we now arrange the ultimate visual results, as it is customary, in three categories, good, moderate, and failure, we have (see page 300):

Ultimate visual acuteness:

Perfect, 96 %, ranging from V $\frac{20}{20}$ to $\frac{20}{200}$.
Moderate, 3 %, " " " $\frac{15}{200}$ to $\frac{5}{200}$.
Failure, 1 %, movements of hand.

The present series emphatically upholds the conclusion I arrived at in the two preceding series, viz.:

Simple extraction is not only the best but also the safest method of removing cataract.

Facta loquuntur.

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[1] These ARCHIVES, vol. xviii., pp. 10 and 11, 1889.

SYSTEMATIC REPORT ON THE PROGRESS OF OPHTHALMOLOGY DURING THE SECOND AND THIRD QUARTERS OF THE YEAR 1889.

By H. MAGNUS, Breslau; C. HORSTMANN, and
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WITH THE CO-OPERATION OF

S. M. Burnett, Washington; Dantone, Rome; Hirschmann, Charkow;
E. Marckwort, Antwerp; P. v. Mittelstädt, Metz; L. Werner,
Dublin; C. H. A. Westhoff, Amsterdam; Schiötz and
Ole Bull, Christiania, etc.

Sections I.–V. Reviewed by Prof. H. MAGNUS.

I.—GENERAL OPHTHALMOLOGICAL LITERATURE.

TEXT-BOOKS, MONOGRAPHS, TREATISES ON GENERAL, BIBLIOGRAPHICAL, AND HISTORICAL SUBJECTS.

193. **Danelius.** Contribution to the ophthalmology of Aëtius. Inaug. Diss., Berlin, 1889. This dissertation contains the text of the vii. volume of Aëtius, which treats of ophthalmology, the *Βιβλια ἰατρικά*, with a translation. The remainder will soon appear.

194. **Hirschberg.** The ophthalmology of the ancient Egyptians. *Deutsche med. Monatschr.*, 1889, No. 38 fl. Presents a review of the ophthalmological knowledge of the Egyptians, with an explanation of the diseases mentioned in the papyrus Ebers, a translation of the chapter of which relating to eye diseases has just been published.

195. **Instituto di clinica oculistica della R. Universita di Napoli.** Anno scolastico, 1887–1888, Paria, 1889. Contains

papers on tuberculosis of the eye, on traumatic changes in the retina, and on capsular cataract, on optical degeneration of Krause's gland, on malignant neoplasm of the caruncle, on operations on the lids, on cataract-extraction, on intra- and extra-ocular cysticerci, on hyaline degeneration in scleral anterior staphyloma.

196. MÄLLER. Text-book of ophthalmology for veterinary surgeons. Stuttgart, 1889.

197. PAPYRUS EBERS. The measures and the chapter on eye-diseases. By Georg Ebers, Leipzig, 1889.

198. SCHMIDT-RIMPLER. Ophthalmology and ophthalmoscopy. For physicians and students. Fourth revised edition. Berlin, 1889. The first edition appeared in 1885; four have therefore appeared in not quite five years.

b.—STATISTICAL PAPERS.

199. BARDE. Hôpital ophtalmique à Genève. Sixth annual report, for the year 1888.

200. ILLINOIS CHARITABLE EYE AND EAR INFIRMARY, Chicago. Sixteenth annual report, 1889. Eye surgeons: Drs. Holmes, Hotz, Ware, Montgomery, and Gardiner.

201. MAGNUS. Report on the ophthalmic clinic of Prof. Magnus at Breslau for the year ending April 1, 1889.

202. SCHIESS-GEMUSEUS. Basel Ophthalmic Institute. Twenty-fifth annual report, for the year 1888. Basel, 1889.

203. SNELLEN. Dentigste Jaaresverslag van het Nederlandsch Gasthuis voor behoeftige en minvermogende ooglijders te Utrecht.

204. TRUC. Bulletin annuel de la clinique ophtalmologique de Montpellier. *Montpellier médic.*, 1889, No. 12; 1889, Nos. 1, 3, 5.

205. WESTHOFF. Kliniek voor Ooglijders. Amsterdam. Erste Jaarverslag voor, 1888. During the first six months 777 patients were treated.

BARDE (199) presents a report on the ophthalmic clinic at Geneva for the last 6 years. Of the 10,414 patients treated, 2,172 were admitted to the clinic. Among 206 extractions there were 12 without iridectomy; in two of the latter there was prolapse of the iris without harmful consequences. Hard and completely ripe cataracts may be extracted according to Barde without iridectomy, or within the capsule. He thinks that the disadvantages of iridectomy have been greatly exaggerated, while the injurious consequences of prolapse of iris and of the secondary cataract, more common as the result of the operation without iridectomy, have been greatly underestimated. MARCKWORT.

During the two years 1887 and 1888 there were treated at the ILLINOIS CHARITABLE EYE AND EAR INFIRMARY (200) 6,398 patients. The important operations were: Extractions according to Graefe, 152; enucleations for various causes, 87; iridectomies, 167; operations on the muscles, 297, 65 of which were advancements. BURNETT.

MAGNUS (201) treated 4,005 patients in his dispensary. Extractions according to v. Graefe, 30, all successful; iridectomies 47; massage of the lens, 8; exenteration, 1; enucleations, 8; squint-operations, 27.

SCHIESS-GEMUSEUS (202) treated 2,162 patients in the dispensary, 540 in the in-door department. Extractions without iridectomy, 36, 34 with good, one with moderate result, one failure. Extractions with iridectomy, 37, 34 with good, two with moderate result, one failure. Two magnet operations, one exenteration, 40 squint-operations, 72 iridectomies.

SNELLEN'S (203) report is mainly devoted to the memory of Prof. Donders, to whom he pays a high tribute as the founder of instruction in ophthalmology, and as founder of the Nederlandsch Gasthuis voor behoeftige en minvermogende ooglijders te Utrecht. In 1888 3,037 patients were treated. WESTHOFF.

II.—GENERAL PATHOLOGY, DIAGNOSIS, AND THERAPEUTICS.

a.—GENERAL THERAPEUTICS.

206. BULL, C. S. The value of electro-therapeutics in lesions of the optic nerve. *N. Y. Med. Jour.*, April 27, 1889.

207. NUEL. Étude expérimentale sur les injections intraoculaires. *Soc. franç. d'opht.*, Aug. 8, 1889.

208. POOLEY, T. R. The uses of an artificial eye-shell for operative and other purposes. *Amer. Jour. Ophth.*, Mar.-Apr., 1889.

209. WIDMARK. The influence of light on the anterior media of the eye. *Nord. med. Arkiv*, 1889, vol. xxi., 1. *Skandinavisches Arch. f. Physiol.*, vol. i. On the influence of light on the skin. *Biol. Fören. Handl.*, Apr., 1889.

In reporting to the N. Y. Academy of Medicine on the therapeutic value of electricity in lesions of the optic nerve, **Bull** (206) says: Up to within the last two years the reporter has employed the galvanic current in every case of optic-nerve lesion that has occurred in his private practice, and in many cases in hospital practice. His results were so negative and in many cases so unfavorable that for two years he has entirely given up galvanism as a method of treatment in these diseases. In traumatic anæsthesia of the optic nerve, without any lesion, however, some good sometimes comes from a careful use of electricity.

Burnett.

Nuel (207) made experiments on the effect of injections upon the parts which line the anterior chamber. Injections of pure water caused a detachment of the endothelium of Descemet's membrane in one fourth to half a minute. The detached shreds fall into the anterior chamber and are absorbed. Injections of solutions of boracic acid are less harmful. Physiological solutions of salt are harmless, and may therefore be recommended to the adherents of injections into the anterior chamber after cataract operations.

Marckwort.

Pooley (208) has used very successfully the glass shield of Brière in keeping the globe and eyelids separated after detachment in symblepharon. No re-adhesions took place, and vision was very much improved. He also suggests the wearing of the shield over the cornea of the healthy eye in case of unilateral blennorrhœa of the conjunctiva.

Burnett.

Widmark (209) reports on the investigations which he made to determine the effect which sunlight and the arc-light may have upon the anterior media of the eye and upon the skin. Sunlight and the light of an electric lamp of 1,200-candle power produced a state of irritation in the eye of a rabbit, which in a marked degree resembled snow-blindness and the electric ophthalmia of man: chemosis and injection of the ocular conjunctiva, contraction of the pupil, change of color of the iris, haziness, and loss of epithelium of the cornea, and moderate secretion of the conjunctiva. The symptoms disappeared after two to four days. The question whether these symptoms are due to direct irritation of the anterior media of the eye, or to a reflex from the blinded retina, are solved in the following manner. A screen was placed before the rabbit's eye, with an aperture of 2 *mm.* Through this aperture and the pupil the light fell upon the retina, the other parts of the eye being protected against the light. In six such experiments the anterior media of the eye remained entirely unaffected. In six other experiments, in which the pupil and the retina were protected against the light, the above-mentioned symptoms, however, appeared, showing that the condition described depends upon the irritation of the anterior media of the eye, and not upon a reflex action from the blinded retina. Glass and quartz crystals are about equally permeable to the ultra-red and luminous rays, but differ from each other by the fact that glass absorbs the ultra-violet rays, while quartz crystals allow them to pass through. This difference was utilized in investigating which rays were the most effective. It was found that the light which had passed through glass, and therefore contained few ultra-violet rays, exerted scarcely any effect, while the light which had passed through the quartz crystal, and therefore contained many ultra-violet rays, produced severe irritation of the anterior media of the eye. Similar effects were produced upon the skin. The ultra-violet rays caused an affection like eczema solare et electricum, an erythema which remained for several days, and was followed by marked desquamation. The experiments also show that the ultra-violet rays possess the power of producing pathological changes upon the surface of the body when acting with sufficient intensity. SCHIÖTZ.

b.—GENERAL DIAGNOSIS.

210. ANGELL. On the use of atropine in the correction of optical defects by glasses. *Jour. Ophth., Otol., and Laryng.*, vol. i., 1889.

211. CHISOLM, J. J. Needless and annoying restraints in eye-surgery. *Jour. Amer. Med. Assoc.*, Aug. 17, 1889.

212. COLLINS. A simple improvement for specimens of eyes mounted for microscopical examination. *Ophth. Rev.*, vol. viii., p. 164.

213. GUILLSBRAND. En praktisk metod at bestämmer hornhinnuns astigmatism genom den s. k. denivelleringen af de oftalmometriska bilderna. *Nord. ophth. Tidsskr.*, vol. ii., No. 2, p. 93.

214. GROENOUW. Where lies the anterior boundary of the ophthalmoscopically visible fundus? *v. Graefe's Arch. f. Ophth.*, vol. xxxv., 3, p. 29.

215. NUEL et LEPLAT. Astigmatisme et ophtalmométrie. *Ann. d'ocul.*, vol. ci., p. 166.

216. PHILIPPS. The electro-magnet. *Jour. Ophth., Otol., and Laryng.*, Jan., 1889.

CHISOLM (211) returns to the discussion of the freedom to be allowed to eye-patients, particularly the subjects of cataract extraction. He does not, as a rule, even put his cataract patients to bed, and allows them the use of the eye not operated on. He, however, closes the lids of the eyes operated on with strips of isinglass plaster. BURNETT.

COLLINS (212) attaches a plano-convex lens to a half-open receptacle which contains the specimen.

GUILLSBRAND (213) reports the following: When a line is regarded through a doubly refracting prism, it is seen double as soon as it makes an angle with the doubling plane of the prism, denivellation. When an astigmatic cornea is examined with an ophthalmometer, with a straight line as object, no denivellation is observed in the two principal meridians, but in all others, and most markedly in those which form an angle of 45° with the principal meridians. In measuring the denivellation the astigmatism can be determined, and so the author has constructed upon this principle a little practical ophthalmometer which is held in the hand like an ophthalmoscope. It is to be had of Collin, Stockholm, price 75 kronen. SCHIÖTZ.

GROENOUW (214) finds that the anterior boundary of the ophthalmoscopically visible fundus lies 8.5 *mm* behind the corneal margin, in myopic eyes a little farther, in hyperopic a little nearer; in aphakial eyes this distance is reduced to 6.5 *mm*. We can see an area of the fundus equal to 770 to 800 times that of the papilla.

PHILIPPS (216) reports on three successful cases of extraction with the magnet. In case of failure he advises immediate enucleation.

c.—GENERAL PATHOLOGY.

217. CHIBRET. Bactériologie ophtalmologique. *Soc. d'opht. de Paris*, April, 1889.

CHIBRET (217) made experiments as to the presence of micrococcus aureus in the conjunctival sac and the injurious effect it may possibly have upon the healing of wounds. He comes to the conclusion that we must always be prepared for the presence of the micrococcus, but that its actual presence is quite compatible with the normal healing of a wound. MARCKWORT.

III.—INSTRUMENTS AND REMEDIES.

a.—REMEDIES.

218. DARIER. De la colchicine en thérapeutique oculaire. *Soc. franç. d'opht.*, Aug., 1889.

219. FELSER. On trichloroiodine. *St. Petersb. med. Wochenschr.*, 1889, No 3. Third Congress of Russian Physicians at St. Petersburg.

220. FOWLER. The use of phytolacca decandra in ophthalmic practice. *Jour. Ophth., Otol., and Laryng.*, Jan., 1889, vol. i.

221. GIBSON and TELKIN. A hitherto unobserved effect of the salicylates. *The Pract.*, 1889, p. 17.

222. KROLL. The curative action of cocaine. *Berliner klin. Wochenschr.*, 1889, No. 32.

223. PAUL. The antiseptic properties of saccharine in ophthalmology. In solutions of 1:250–500. *Deutsche med. Ztg.*, 1889, No. 2.

224. DE VRIESE. L'éphédrine et la pseudo-éphédrine. Nouveaux mydriatiques. *Ann. d'ocul.*, vol. ci., p. 182.

In obstinate cases of arthritic affections of the eye DARIER (218) recommends colchicine in granules of 2 mgrm., with an increase of the dose to 6 mgrm. It must however be discontinued should the intestinal canal become affected. Brunschwig saw disagreeable consequences after doses of ½ mgrm. (According to Ewald's pharmacopœia the dose should be 0.0006–0.003, one to three times daily.—Rev.) MARCKWORT.

FELSER (219) highly recommends trichloroiodine (ICl_3) as an antiseptic.

GIBSON (221) observed marked mydriasis with loss of reaction to light after the use of salicylate of sodium in doses of 1.2 grms, every two hours, eight hours after the first dose had been taken. The symptoms disappeared thirty hours after the last dose.

KROLL (222) recommends cocaine in five per cent. solutions in scrofulous blepharospasm, in episcleritis and iritis.

DE VRIESE (224) reports at length on experiments with ephedrine (derived from ephedra vulgaris varietas helvetica) and pseudo-ephedrine. These two alkaloids dilate the pupil, but do not paralyze the accommodation. As their effect disappears more rapidly than that of homatropine, these mydriatics are to be preferred to the latter for ophthalmoscopic examinations. Although ephedrine must be used in ten per cent. solutions its use is cheaper than that of homatropine. MARCKWORT.

b.—INSTRUMENTS.

225. DUANE, ALEXANDER. Some new tests for insufficiencies of the ocular muscles, together with a system of abbreviations suitable for note-taking. *N. Y. Med. Jour.*, Aug. 3, 1889.

226. LANDOLT. Objets types. Black letters on porcelain. *Soc. franç. d'opht.*, Aug. 9, 1889.

227. STAERR. A new (?) optometer. *Amer. Jour. Ophth.*, Mar.–Apr., 1889.

The tests described by DUANE (225) are not exactly new, but a new importance is attached to some old ones. The "screen test" for ocular muscular insufficiencies is modified, so that instead of watching the amount of deviation of the eye under the screen, the apparent movement of an object, a candle for instance, when the screen is removed, is noted. If there is insufficiency the image is shifted when the eyes are covered alternately, forming a sort of parallax, and the direction of the movement gives the muscles or set of muscles affected. The author claims great delicacy for this test. In testing for abduction and adduction he uses a small point which is moved from the centre to the periphery (right and left) of the field of fixation, and the place noted at which diplopia sets in. This is more readily accomplished by having the point stationary, and turning the head on its vertical axis. The table of abbreviations is quite lengthy, and most of them are well chosen. BURNETT.

IV.—ANATOMY.

a.—MALFORMATIONS AND EMBRYOLOGY.

228. GUNN. Congenital malformations of the eyeball and its appendages. *Ophth. Rev.*, July, 1889.

229. RANKE. The Mongolian eye as a temporary state in German children. *Neurol. Centralbl.*, 1889, No. 5; *Centralbl. f. prakt. Augenhk.*, 1889, p. 273.

GUNN (228) discusses the development of monolateral and bilateral anophthalmus, of cyclopia, cryptophthalmus, microphthalmus, buphthalmus, coloboma of the lids, and ptosis. WERNER.

Ranke (229) finds that the principal peculiarity of the Mongolian eye consists in a certain degree of epicanthus. In children of the Caucasian race a similar condition is observed, but it disappears in the course of years.

b.—SCLERA, CORNEA, IRIS, LENS, RETINA, OPTIC NERVE.

230. **Leroy.** Sur la forme de la cornée normale. *Acad. des Sciences. Sem. méd.*, 1888, pp. 42 and 45.

231. **Tornatola.** Direzione delle fibre dei nervi ottici nel chiasma. *Ann. di ottalm.*, vol. xviii., 3, p. 229.

Leroy (230) made accurate measurements of the surface of the normal cornea with his ophthalmometer and found that the vertical meridian had the same curvature in the upper and lower half, while in the horizontal meridian the curvature of the outer was different from that of the inner half. The curvature of the cornea depends to a great extent upon the ocular muscles.

MARCKWORT.

According to **Tornatola** (231), who has carefully examined three chiasmas in which one optic nerve was atrophic, distinct semi-decussation exists in man. Experiments on animals showed that in rabbits complete decussation takes place, while in dogs two thirds of the fibres of the optic nerve pass into the tractus of the other side, one third remaining on the same side. DANTONE.

V.—PHYSIOLOGY.

a.—RETINA AND CENTRAL ORGAN.

232. **Ljubinski.** On the influence of the electric light on the eyes of those engaged with it. *St. Petersb. med. Wochenschr.*, 1889, No. 3. Third Congress of Russian Physicians at St. Petersburg. *Centralbl. f. prakt. Augenhk.*, 1889, p. 176.

Ljubinski (232) found the continued action of the electric light produces attacks of pain, also lachrymation and photophobia, symptoms which mainly manifest themselves at night. The ophthalmoscope shows hyperæmia of the optic nerve ; at the macula small plaques are observed.

b.—CORNEA, IRIS, MUSCLES.

233. **Ahrens, R.**, and **Ahrens, A.** Further experiments on anisomorphous accommodation. *Zeh. klin. Monatsbl. f. Augenhk.*, 1849, p. 291.

234. **Catania, Romano.** Sur le striscie di filtrazione della cornea e dell' iride. *Boll d'ocul.*, vol. xi., 12–13, p. 89.

Ahrens (233) shows that the youthful eye is capable of obtaining clear images through cylindrical glasses, though the effort be continued through several minutes and gradually increased. This power is probably due to unequal muscular action at different points of the margin of the lens and consequent anisomorphous change in the lens.

Romano Catania (234), after numerous experiments on the filtration of colored liquids, which were injected into the anterior chamber, into the vitreous, or under the skin, has come to the following conclusions: (1) There are so-called filtration-bands in the cornea, which permit the aqueous humor to pass through, as Knies was the first to show; (2) the epithelium of Descemet's membrane permits of the passage of liquids, which however it checks and regulates; (3) in the iris no such bands exist, as Ulrich has assumed, the aqueous humor passing into the anterior chamber only through the pupil. DANTONE.

c.—COLOR-PERCEPTION.

235. **Ssamujlow.** The question of the exhaustion of the retina by different colors (red, green, blue). *St. Petersb. med. Wochenschr.*, 1889, No. 3. Third Congress of Russian Physicians at St. Petersburg.

According to **Ssamujlow** (235) the retina is most rapidly exhausted by red, less by green, still less by blue.

Sections VI.–XI. Reviewed by Prof. HORSTMANN.

VI.—ANOMALIES OF REFRACTION AND ACCOMMODATION.

236. **Bull.** Les variations de l'astigmatisme avec l'âge. *Soc. franç. d'opht.*, Aug. 8, 1889. The astigmatism does not change materially, either in childhood or in more advanced age.

237. **v. Hippel.** The influence of hygienic measures on school-myopia. Giessen, 1889.

238. **Kessler, H. M. C.** Papilla falcata en papilla annullata. *Weekblad van het Nederlandsch Tijdschrift voor Geneeskunde*, 1889, Deel ii., p. 329.

239. **Leroy.** De la polyopie monoculaire. *Soc. franç. d'opht.*, Aug. 8, 1889.

240. MOTAIS. De l'hérédité de la myopie. *Arch. d'opht.*, 1889, No. 4, p. 321.

241. RANNEY, A. L. Eye-strain as a cause of headache and neuralgia. *N. Y. Med. Rec.*, June 22, 1889.

242. SCHMIDT-RIMPLER, H. Remarks on Stilling's paper; measurements of the orbit in different refractive conditions. *Fortschritte d. Med.*, 1889, No. 15.

243. SCHMIDT-RIMPLER, H. Once more the measurements of the orbit in different refractive conditions. *Fortschritte d. Med.*, 1889, No. 20.

244. SCIMENI. Sull' astigmatismo corneale in seguito ad estrazione di cateratta. *Ann. di ottalm.*, vol. xviii., 1–2, p. 147.

245. STILLING. Further orbital measurements of myopic and normal eyes. *Fortschritte d. Med.*, 1889, No. 17.

In the course of the last nine years v. HIPPEL (237) has regularly examined the eyes of the scholars of the high school at Giessen, and finds that a not inconsiderable part becomes myopic, while in others the already existing myopia increases, in spite of most perfect architectural construction and model interior equipments, in spite of the prevention of overburdening with work and regular medical control. He finds the cause on one hand in near-work *per se*, on the other in the unfavorable hygienic influences which act in the house upon the eyes of the children. The former cause of myopia can never be removed, unless we give up the fruits of modern civilization.

Stilling's investigations have shown that the terms "atrophy" and "conus" are not correct in myopia. KESSLER (238) therefore proposes a term based upon what one sees with the ophthalmoscope, *i. e.*, a papilla with a crescent (falx) and a papilla with a ring (annulus). At the Utrecht clinic the expressions "papilla falcata" and "papilla annullata" are used. For staphyloma posticum and sclerotico-choroiditis postica he would substitute ectasia (not sclerectasia) postica myopica. WESTHOFF.

LEROY (239) reports a case of monocular polyopia. Valude observed monocular polyopia in himself, and explains it from his myopia. Kalt made the same observation upon himself; he is emmetropic in one eye and has slight myopic astigmatism in the other. Kalt and Boucheron explain monocular polyopia by spasm of accommodation. MARCKWORT.

In order to study the influence of heredity upon the development of myopia, Motais (240) was not satisfied with making inquiries as to the vision of the relatives of his myopic patients, but examined the families themselves of 224 myopic boys and 106 girls, 330 families in all, including grandparents, parents, brothers and sisters, etc., in all 1,991 persons. After presenting a series of statistical tables Motais comes to the following conclusions: the heredity of myopia has been firmly established, and was found in 216 cases among 330 families (65 %). It descended from the father to the daughters in 86 %, from the mother to the sons in 79 %. When both parents were myopic the myopia descended with equal frequency to the sons and daughters. In myopia of the grandparents this crossing of the heredity was not so apparent. Omission of one generation was also observed: grandmother myopic, 2 sons not, 4 grandsons myopic, 4 grandchildren not; grandfather myopic, 3 daughters not, 4 among 5 granddaughters myopic. Myopia, when hereditary, appears at an earlier age, develops more rapidly, attains a higher degree, and is more frequently attended with intraocular changes than acquired myopia. Heredity is favored by taxing the eyes under unhygienic conditions (51 % instructed against 25 % uninstructed children of myopic families), by astigmatism (14 % above 0.75 D), and by flattening of the orbit (30 %). v. Mittelstädt.

In this paper Ranney (241) again takes up the subject of eye-strain as a cause of headache, and tabulates fifty consecutive cases of headache and neuralgia treated exclusively for the correction of eye-strains. Of course great attention is given to the disturbance in the equilibrium of the external muscles or "heterophoria," since the author is a firm believer in this as a cause of various pathological nervous conditions. The histories of all the fifty tabulated cases are given in greater or less detail, and seem to bear out the claims of the author as to the efficacy of the treatment, whether by glasses or by graduated tenotomies of the muscles. Burnett.

Schmidt-Rimpler (242) opposes the hypothesis of Stilling, that a low, broad orbit can be the cause of the development of myopia. According to his measurements and investigations, the school-myopia in cases of low orbit had not increased more in three and a half years than in cases of high orbit. In the majority of cases it is impossible to determine from the shape of the orbit whether a scholar in the lower classes will become myopic or not in the course of his studies.

SCIMENI (244) concludes from numerous measurements of the cornea before and after cataract extractions that the operation can cause astigmatism or increase it if it already exists. In one case, which he particularly mentions, the error had increased from 1 D to 18 D. He believes that the cause of the change of curvature is to be found, not only in the manner of healing of the wound, as has thus far always been assumed, but also and particularly in the pressure which the internal and external rectus muscles exert upon the globe, which on account of the escape of the aqueous has been partially deprived of its power of resistance. This is the reason why the greater curvature is found in the meridian parallel to the section. DANTONE.

STILLING (245) presents the results of orbital measurements of 810 persons, mostly students; they confirm his opinion previously expressed, that a low, broad orbit predisposes to myopia.

VII.—LIDS.

246. CERESETO. Ectropion della palpebra inferiore. Nuova applicazione di un vecchio metodo operatorio. *Boll. d'ocul.*, vol. ix., 9, p. 68; and *Gaz. med. di Torino*, vol. xl., 15, p. 337.

247. CHEVALLEREAU. Corps étranger ayant sejourné dix ans dans paupière supérieure. *Soc. d'opht. de Paris*, April 2, 1889.

248. LAGRANGE, F. Anatomie pathologique et pathogénie du chalazion. *Arch. d'opht.*, 1889, No. 3, p. 226.

249. LESZYNSKY, W. M. Bilateral blepharospasm, with divergent strabismus, cured after division of the external rectus muscle. Subsequent restoration of stereoscopic vision. *N. Y. Med. Jour.*, May 18, 1889.

250. MILLINGEN, E. VAN. Remarks on one hundred cases of trichiasis, operated on according to my method of so-called tarsachelioplasty. *Centrabl. f. prakt. Augenhk.*, 1889, p. 193.

251. NOYES, H. D. An operation for some extreme cases of ectropium of the upper lid. *Amer. Jour. Ophth.*, 1889, No. 8, p. 245.

252. PEUNOW, A. J. Two cases of transplantation of excised pieces of skin in plastic operations on the lids. *Sitzungs-Protokoll d. Kaukas. med. Gesellsch.*, vol. xxvi., No. 2.

253. RUTTLE, R. Rare form of ankyloblepharon. *Brit. Med. Jour.*, Sept. 14, 1889, p. 599.

254. SCIMENI. Sulla margino-plastica palpebrale nella trichiasi e nell' entropio. *Boll. d'ocul.*, vol. xi., 12–13, p. 85.

255. SPANGENBERG, A. A case of tuberculosis of the lids and eye. *Inaug. Diss.*, Marburg, 1889.

256. STELLWAG VON CARION. Review of the attempts at transplantation in ophthalmology, and a new case of transplantation of mucous membrane. *Allgem. Wiener med. Ztg.*, 1889, Nos. 27, 28, and 29.

257. ST. VALUDE. Du blepharospasme. Etiologie et traitement. *Arch. d'opht.*, 1889, No. 3, p. 273.

258. VALUDE, E. De la restauration des paupières, désavantages de la greffe cutanée. *Arch. d'opht.*, 1889, No. 4, p. 289; No. 5, p. 394.

259. VAN DUYSE. Sarcome de la paupière. *Ann. d'ocul.*, vol. ci., p. 227.

260. VIGNES. Epicanthus héréditaire. *Soc. d'opht. de Paris*, June 4, 1889.

In an operation for ectropium, CERESETO (246) took a flap from the inner side of the upper arm to cover the large defect, as the old Italian surgeons did in rhinoplasty, and was well satisfied with the result. DANTONE.

CHEVALLEREAU (247) removed from the left upper lid of a patient a piece of glass which had been concealed therein for ten years without causing any irritation, the patient himself not being aware that a foreign body had remained behind after the injury. A month before its removal he had had frequent headaches. At the same time a little elevation appeared in the skin just above the external canthus; at this point Chevallereau extracted the triangular piece of glass, one side measuring 10 *mm*, the other two 23 *mm*. MARCKWORT.

LAGRANGE (248) examined fifteen chalazia, some of them removed with the fibrous capsule and the adjacent cartilage. He assumes three stages of development: retention of the contents of the Meibomian glands, adenitis, and periadenitis, with destruction of the cartilage and development of the tumor towards the conjunctiva or the outer surface of the lid. The contents consist of granulation tissue and broken-down epithelium; the latter contains micrococci, which however play only a secondary part. He did not find any giant-cells. In most cases there was a fibrous capsule, sometimes, especially in fresh cases, it was wanting.

v. MITTELSTÄDT.

LESZYNSKY'S (249) patient, a boy of fourteen, was affected with bilateral clonic blepharospasm, since an attack of scarlatina when six years old, which left him with a divergent strabismus of the left eye. Left eye myopia $\frac{1}{7}$, and astigmatism $\frac{1}{60}$. V = $\frac{20}{40}$—. The left external rectus was divided, and the ametropia corrected. Binocular vision was restored. BURNETT.

MILLINGEN (250) found that after the performance of the tarso-cheiloplasty devised by him the pannus cleared up, and the superficial ulcers accompanying cicatricial trachoma rapidly healed.

In cases where the upper lid is so contracted that there is undue pressure on the eyeball, NOYES (251) cuts through the conjunctiva, tarsus, and orbicularis from the free to the upper edge of the tarsus, leaving the bridge of skin above the wound. The wound is allowed to heal without interference.

BURNETT.

PEUNOW (252) covered the defects caused by the excision of a tumor and a cicatrix with pieces of skin, 3 *cm* wide, which in one case he took from the forearm of the patient himself, in the second from the forearm of the son of the patient, the pieces being of the whole thickness of the skin. The results were very satisfactory. Peunow ascribes the success (1) to the strict antisepsis of the bandage and the avoidance of strong antiseptics in manipulating the flap and the defect; (2) to the transfer of the pieces in their entire thickness; (3) to the careful freshening of the edges and surface of the defect; (4) to the accurate adaptation of the picce of skin; and (5) to the complete arrest of bleeding. HIRSCHMANN.

RUTTLE (253) observed a case in which the lids of both eyes had been congenitally united at the inner side by an elastic, skin-like membrane. They were divided without difficulty.

WERNER.

The method of operating for entropium in cases of highly degenerated tarsus proposed by SCIMENI (254) is a modification of that of Burchardt. After dividing the edge of the lid and forming a rectangle in the skin of the lid the short sides of this rectangle are not detached at the two canthi, but the whole strip of skin is drawn across the ciliary margin and inserted in the intermarginal space, the ciliary margin having been previously detached at the outer ends and attached with sutures to the upper edge of the incision. The strip is maintained in position by a bandage without the aid of sutures. In twelve among fifteen cases operated on according to this method the bridge of skin became attached in its whole extent, in three a small part sloughed away in the middle of the lid and was replaced by cicatricial tissue.

DANTONE.

SPANGENBERG (255) observed a case of tuberculosis of the lids in a boy aged fourteen. The lids of the right eye were reddened, infiltrated with nodules, and ulcerous ; the eyeball had been converted into a nodular, brownish-red tumor. The diseased parts of the lids and the eyeball were removed and proved to be tuberculous. The disease first appeared in the skin of the lids, whence it spread to the conjunctiva, cornea, sclera, and inner parts of the eye. The microscope and cultures demonstrated the existence of tuberculosis. Tubercle bacilli were shown to be present.

ST. VALUDE (257) briefly discusses the causes and treatment of the usual forms of blepharospasm, and calls attention to the fact that not only hysteria, but also forms of psychic disease can be the cause. He saw a man who had suffered for eight years with clonic blepharospasm, who, though otherwise healthy, was affected with hereditary taint on both the father's and mother's side, and who had stuttered since youth and was always troubled without cause about his future. At one time the orbicularis was divided, but without success. The author warns against operative procedures in cases of purely nervous blepharospasm, as they are absolutely useless. They only come into consideration when the disease is of a distinct reflex nature. v. MITTELSTÄDT.

After a brief discussion of the various methods of blepharoplasty VALUDE (258) expresses himself against the transplantation of flaps without a pedicle as a general method. He thinks it is applicable only in a very limited number of cases. He shows in two cases of cicatricial ectropium after burns, that although the flaps had taken hold, they completely disappeared after a few months by absorption, making the operation a failure, while in another case a very good result was obtained with a pedunculated flap which had been transplanted to a similarly injured lid. He presents a review of seventy-five cases of transplantation of a flap without a pedicle, the results of which he thinks were published too soon, and yet only fifteen among them were successful, indicating that this method is not generally applicable. According to Valude the method of operation should be chosen on the following principles : 1. When the scars are confined to the lids and cause partial or complete ectropium, pedunculated or sliding flaps should be chosen. He then describes the different procedures for the various kinds of ectropium. 2. When the lids and neighborhood consist of non-adherent cicatricial tissue, flaps without a pedicle are not advisable ; the best results are obtained with pedunculated flaps when they consist of vascular cicatricial tissue ; in low degrees a sliding flap is sufficient. 3. When the ectropium is surrounded by cicatricial tissue which cannot be used for plastic purposes, flaps without a pedicle may be tried, though the result is uncertain. The old Italian method, formerly used in rhinoplasty, is well suited for this purpose, when the patient is willing and the operator is familiar with the method. 4. In cases in which the defect in the lid is not due to ectropium, but to the removal of a cancer, flaps with or without a pedicle may be employed as no retraction of the surrounding tissue is to be feared ; the method of Thiersch, especially, is to be commended. It is very easy and well adapted for cases in which the defect is situated at one of the canthi (report of a case). In cases of cancer the wound may also be covered when it begins to granulate, a procedure well suited for the observation of relapses. The defect need not be covered for a long time, especially when it is a large one, as the neighboring skin will partly cover the defect spontaneously, thus making a less extensive secondary plastic operation necessary, as shown by a case of Verneuil reported by the author.

V. MITTELSTÄDT.

VAN DUYSE (259) presents a few more details in regard to a previously published case of sarcoma of the lid. The prognosis of this disease is very bad. An early and extensive operation must be performed, so that at least the whole lid must be sacrificed.

MARCKWORT.

VIGNES (260) operated on a man aged thirty-six for bilateral epicanthus, complicated with narrow palpebral fissure, ptosis, and pyramidal cataract. Epicanthus of more or less marked degree was observed in many members of the family. As early as 1845 Sichel found epicanthus and ptosis in the grandfather of the patient. Vignes observed the same abnormity in the man's children.

MARCKWORT.

VIII.—LACHRYMAL APPARATUS.

261. NOYES. Entrance of an eyelash into the inferior canaliculus. *Am. Jour. Ophth.*, Aug., 1889.

262. SGROSSO. Sul cilindroma e sul cancro melanico della caruncula lagrimale. *Ann. di ottalm.*, vol. xviii., 1–2, p. 57.

263. Westhoff. Adeno-carcinoma glandulæ lagrymalis. *Kliniek voor Ooglijders*, 1889, p. 8.

Noyes (261) found a fine eyelash with its point sticking in the lower canaliculus and its body rubbing against the eyeball and giving rise to much irritation. The reviewer has seen one such case. Burnett.

Sgrosso (262) describes two small tumors of the caruncle, one of which proved to be a cylindroma, the other a melanotic sarcoma. According to his statement no case of cylindroma of the caruncle has thus far been observed. Dantone.

Westhoff (263) removed the lachrymal gland, which was larger than a hen's egg, from a woman aged sixty-three, with preservation of vision. Twenty-five years ago the left eye received a blow, and since that time it had protruded more and more, so that now it stands 10 *mm* lower and 20 *mm* farther forward than the right. The eyeball is freely movable in every direction. The choroid around the optic nerve is markedly atrophic from the tension on the nerve. Westhoff.

IX.—MUSCLES AND NERVES.

264. Beard, C. H. Advancement with but a single suture. *Amer. Jour. Ophth.*, March–April, 1889.

265. Gast, R. A. A case of bilateral congenital ophthalmoplegia. *Zeh. klin. Monatsbl.*, vol. xxvii., p. 214.

266. Günsburg, F. Report of cases of congenital anomalies of the muscles. *Zeh. klin. Monatsbl.*,vol. xxvii., p. 263.

267. Hubbell, A. A. Cases of ocular paralysis. *Buffalo Med and. Surg. Jour.*, Sept., 1889.

268. Landmann, O. On cycloplegia post-diphtheritica. *Amer. Jour. Ophth.*, May, 1889.

269. Müller, C. E. Contribution to the etiology and operation of convergent strabismus. *Inaug. Diss.*, Marburg, 1889.

270. Peters, A. On paralysis of convergence. *Centralbl. f. prakt. Augenhk.*, 1889, p. 225.

271. Wright, J. W. Rheumatism of the ocular muscles. *N. Y. Med. Rec.*, Sept. 28, 1889.

Beard's (264) modification of the advancement operation is as follows: The conjunctiva is dissected off the muscle, and the latter is loosened from its attachment to the eyeball up to its insertion near the cornea. A thread is armed with two needles, one of which is passed through the upper edge of the tendon, the other through the lower edge, thus leaving a loop of thread on the upper side of the tendon. The tendon is then cut about 2 *mm* in front of the stitch. The stump of tendon remaining is then cut off close to the sclera. The upper needle is then run through and under the conjunctiva to a point above the vertical diameter of the cornea, and the lower thread is run similarly below. One of the needles is then brought back and passed through the loop of the thread on the top of the tendon, and the two ends are drawn until the tendon is advanced to the desired point and then tied. BURNETT.

GAST (265) observed in a woman aged thirty-three complete immobility of both eyeballs, with normal power of accommodation and almost normal sight.

GÜNSBURG (266) reports the complete absence of any action of the external rectus in a grandmother, father, and two daughters, while a third daughter had alternating divergent strabismus.

HUBBELL (267) reports a case of paralysis of both facial and both abducent nerves following injury. After discussing the various causes that might lead to such a result, he concludes that it can be best accounted for on the supposition of a hemorrhage near the median line of the floor of the fourth ventricle between and affecting the nuclei of the abducent and the nuclei of the facial nerves. He also relates the history of a case of total monocular ophthalmoplegia externa and interna also from injury (the "punch" of an umbrella-stick in the eye). Vision was entirely lost, the optic disc was pale, and the retinal vessels were slightly reduced in size. BURNETT.

LANDMANN (268) thinks that in the majority of cases of cycloplegia sine iridoplegia the paralysis is due to the perverted state of the blood influencing the nutrition of the muscle and not the nerve-centre or nerve-endings. BURNETT.

MÜLLER (269) has compiled 154 cases of convergent strabismus from the material of the Marburg ophthalmic clinic, arranging them according to age and refraction. In seventy-eight per cent. there was H in both or at least one eye, in twelve per cent. there was E in both, in five per cent. M in both ; he calls attention, however, to the fact that even in cases in which at present M or E exists there may have been H at the time the strabismus developed. He also reports cases of spontaneous cure of strabismus, the H persisting. The primary effect of the operation must be measured very carefully ; subsequent exercises with the stereoscope and stimulation of the "desire of binocular fixation," as Schmidt-Rimpler expresses it, are very important. Of seventy-seven patients with divergent strabismus sixty-four per cent had myopia in one or both eyes.

PETERS (270) reports two cases of paralysis of convergence, the one the consequence of an injury, the cause of the other unknown. The subjective symptoms consisted principally in dizziness and diplopia. When an object in the median line is intently regarded, the pupil does not respond, and the eyeballs remain immovable. The affection is of central origin.

WRIGHT (271) thinks that many of the intractable head-pains from which so many suffer, are due to some form of rheumatism of the ocular muscles, and that it should be looked for in such cases. BURNETT.

X.—ORBIT AND NEIGHBORING CAVITIES.

272. ALT, A. Orbital sarcoma in children. *Amer. Jour. Opht.*, Feb., 1889.

273. AHRENS, R. Another case of cavernous tumor of the orbit. *Zeh. klin. Monatsbl. f. Augenhk.*, vol. xxvii., p. 416.

274. BENSON. Aneurism in the right orbit cured by digital compression. *Br. Med. Jour.*, 1889, p. 716.

275. BRUNSCHWIG. Angiome caverneux encapsulé de l'orbite. *Arch. d'opht.*, 1889, No. 5, p. 419.

276. HALTENHOFF. Exophtalmie congénitale syphilitique. *Ann. d'ocul.*, vol. cii., p. 108.

277. JEAFFRESON, C. S. A record of sixteen cases of orbital tumor. *Lancet*, July 20, 1889, p. 110.

278. VALUDE. Kyste hydatique de l'orbite. *Rec. d'opht.*, 1889, No. 8, p. 486.

ALT (272) reports two cases of sarcoma of the orbit in children on whom he operated. In the first, the mother had a sarcoma of the axilla, and other members of the family had died of cancer. An attempt was made to enucleate the tumor and save the eyeball, but it was not successful. The tumor, which was the size of a hazel-nut, grew from the main wall of the orbit. The child was one and a half years old. A few days after the operation the child died of erysipelas. The tumor was found to be a round-celled sarcoma. Second case: boy aged two years. The whole contents of the orbit were removed. The tumor, which was a round-celled sarcoma with portions of a myxomatous character, occupied the outer upper portion of the orbit. There was a return of the growth in six weeks, and as there were cerebral symptoms nothing further was undertaken. BURNETT.

The case of AHRENS (273) was one of cavernoma of the orbit. The tumor was 2 *cm* long and 11 *mm* thick, and was removed, with preservation of the globe.

BENSON (274) relates a case of orbital aneurism in a man aged thirty-eight. The disease began two months after a blow with a stick. No thrill could be felt, nor was there any visible pulsation of the globe. At the outer side of the upper cul-de-sac of the conjunctiva there was a collection of dark, tortuous veins, and the veins in the retina were darker and fuller than those in the other eye. The patient was cured by digital compression of the carotid. WERNER.

BRUNSCHWIG (275) removed from an otherwise healthy woman of thirty-two, a cavernous angioma which had developed in the left orbit in the course of eight years. When first examined there was only moderate exophthalmus of the left eye, but no diagnosis could be made, as all the symptoms characteristic of a cavernous tumor were absent. As there were no indications of a malignant growth, a lipoma was thought to be the cause. Five years later the eye protruded still more, and although a soft, incompressible tumor could be felt in the upper inner part of the orbit, an exact diagnosis was impossible. Mobility perfect, no limitation of the visual field, no diplopia, no pain. An incision was made at the lower outer margin of the orbit, and the tumor removed. It proved to be an encapsuled cavernoma, which had developed outside the funnel of muscles. Sight and the ophthalmoscopic condition, which had not been materially affected before the operation, remained unchanged, and only paralysis of the superior oblique resulted, which may necessitate an operation. Two illustrations show the condition of the patient before and after the operation. v. MITTELSTÄDT.

HALTENHOFF (276) observed marked exophthalmus in a newborn child, which he ascribed to a gumma of the orbit. The eyeball protruded about 1 *cm*, and deviated slightly downward and outward. In endeavoring to reduce it a firm resistance was encountered. Inunctions with blue ointment were made twice daily, and iodide of potassium was given internally, whereupon the exophthalmus completely disappeared. In an older sister congenital syphilis was undoubtedly shown to exist. MARCKWORT.

JEAFFRESON (277) records 16 cases illustrating the following classes of orbital growths : (1) growths originating in the orbital tissue unconnected with the eyeball; (2) those arising from the periosteum or bony walls ; (3) those commencing in the neighboring cavities ; and (4) those due to vascular disease in the cavity of the orbit and closely adjoining part of the cranium. Among the latter was a case of teleangiectasia of the orbit, which was removed successfully along with the globe in spite of alarming hemorrhage ; also a blood-cyst of the orbit, which occurred during a fit of whooping-cough, and was cured by puncture with trocar ; and finally a pulsating tumor, for which the common carotid was ligated with fatal result. WERNER.

VALUDE (278) found during an operation an echinococcus-cyst of the orbit instead of the dermoid cyst he had expected. It contained much chloride of sodium, but no hooks could be found. The sac was easily removed by traction with the forceps. Healing by first intention. Valude advises always to remove such cysts. Meyer remarks that these cysts are usually much larger than the one operated on by Valude ; the patients, however, generally do not present themselves until the cysts have begun to suppurate. MARCKWORT.

XI.—CONJUNCTIVA, CORNEA, SCLERA, ANTERIOR CHAMBER.

279. ABADIE. Symblépharon rébelle guéri par transplantation d'un lambeau de muqueuse buccale. *Soc. d'opht. de Paris*, April 2, 1889.

280. ALT, A. On pterygium. *Amer. Jour. Ophth.*, March–April, 1889.

281. BASEVI. Il micrococco della congiuntivite folliculare. *Ann d'ocul.*, vol. ci., p. 159.

282. BURCHARDT. The galvano-caustic treatment of follicular conjunctivitis. *Deutsche militärärztl. Zeitschr.*, 1889, No. 4.

283. GASPARINI. Innesto di congiuntiva e cornea di cane in un caso di simblefaron con attecchimento immediato e successiva atrofia. *Ann. di ottalm.*, vol., xviii., 3, p. 204.

284. GIOTTI. Contributo alla cura della congiuntivite granulosa. *Boll. de scienze med.*, vol. xl., 1, p. 21.

285. HOWE, LUCIEN. The purulent conjunctivitis of infants, and blindness in the State of New York. *N. Y. Med. Jour.*, May 4, 1889.

286. NUEL et LEPLAT. Conjonctivite folliculaire. *Ann. d'ocul.*, vol. ci., p. 159.

287. PECHDO. Phlegmon spontané de l'œil ; resorption purulente. *Soc. franç. d'opht.*, August 13, 1889.

288. PFALZ, G. The operative treatment of follicular conjunctivitis. Bonn, 1889.

289. SCHRÖDER, TH. The surgical treatment of chronic follicular trachoma. Results of removing the granulations with a metallic brush. *Wratsch*, 1889, No. 34.

290. SWART, ABRAHAMSZ. Restoration of the conjunctiva with scales of epidermis. *Kliniek voor Ooglijders*, 1889, p. 12.

291. TOSI. Cura della congiuntivite granulosa colle scarificazioni associate alla cauterizzatione. *Giorn. med. del R. Esercito e Marina*, vol. xxxvii., 4, p. 363.

292. TROUSSEAU. Lupus et tuberculose oculaire. *Soc. franç d'opht.*, August 8, 1889.

293. VOSSIUS. A. The practical value of the operative treatment of follicular conjunctivitis. *Therap. Monatsh.*, 1889, Nos. 6, 7.

In a case of symblepharon ABADIE (279) successfully transplanted mucous membrane from the lips. The eye had been destroyed by combustion with lime, and the stump had become united with the lower lid in such a manner as to obliterate the lower conjunctival sac and make the wearing of an artificial eye impossible. Abadie took a flap ½ *cm* long and 1 *cm* wide from the inner surface of the upper lip and covered the defect on the inner surface of the lid with it. MARCKWORT.

ALT (280) thinks that for the formation of pterygium we must have first a loss of substance at the corneal margin, secondly, a microbe which grows slowly into the corneal tissue, and thirdly, a small fold of conjunctiva dragged upon the cornea by the continued progress of microscopical death and attempted repair in the corneal tissue. The operation he prefers is that of excision, drawing the dissected conjunctiva over the wound and cauterizing the cut surface with pure carbolic acid. The after-treatment is antiseptic. BURNETT.

Basevi (281) has obtained from cultures of the secretion of follicular conjunctivitis on gelatine and agar-agar a very small, round coccus, with motion of its own, which differs materially from the diplococci of trachoma and conjunctival catarrh. When introduced under the conjunctiva of animals (monkeys, dogs, rabbits, pigeons), it produces a condition very similar to that of follicular conjunctivitis in man. Dantone.

Burchardt (282) employed the galvano-cautery in follicular conjunctivitis with good result. He destroyed from 30 to 40 follicles at one sitting, and repeated this after 5 or 6 days. The patients were cured in from 3 to 4 weeks.

Gasparini (283) describes an operation for symblepharon due to combustion with lime. After the adhesions had been divided the wound was covered with conjunctiva and a small piece of cornea taken from a hunting-dog.

Giotti (284) reports that in 30 cases of trachoma he tried the scarifications recommended by Abadie and followed by cauterization with sulphate of copper in glycerine (1:8) with excellent result. Dantone.

Howe (285) finds, by an examination of the statistics, that blindness in the State of New York (as well as in other parts of the country) is increasing nearly eight times as rapidly in proportion as the population. More than 20 % of this blindness is caused by the purulent conjunctivitis of infants. He thinks the State should interfere and control the management of such cases in their incipiency, by legislation, as is done in some countries in Europe. He adopts the Credé method, or some modification of it, as the most efficient. Burnett.

In trachoma Pfalz (288) recommends excision of both cul-de-sacs with a strip of tarsus. He incises the fornix close to the canthus and separates it from the ocular and palpebral conjunctiva close to the edge of the tarsus. He then cuts it out as far as the opposite canthus. The width of the piece of mucous membrane thus excised varies between 5 and 8 *mm.* The operation is done both upon the upper and lower lid. The edges of the wound are united with sutures. The result in 70 eyes thus treated was satisfactory.

Schröder (289) uses a brush of nickelled-silvered copper wire, with which he removes all the visible trachoma follicles from the conjunctiva, and even the pannous cornea, under cocaine, and in one sitting, if possible. He finds this method more satisfactory and easier than other surgical procedures, and suitable even for out-door patients. The wounds caused by the instrument heal rapidly and easily, leaving only smooth scars. The patients are improved. In some cases, however, he observed relapses.

HIRSCHMANN.

SWART ABRAHAMSZ (290) observed a man into whose eye a piece of glowing iron had entered 16 years ago, causing symblepharon. After it had been divided, the upper lid was everted, and the ciliary margin attached to the skin of the brow with three sutures. He then cleansed the wound and waited until the bleeding had ceased. Then little scales of epidermis, which had previously been abscised from the arm and placed in 0.6 % sterilized solution of salt, were laid like shingles upon the wound and both eyes bandaged. Several scales took hold. In a second operation a few scales were transplanted upon the ocular conjunctiva. The result of these two operations was very satisfactory.

WESTHOFF.

TOSI (291) remembers that the scarifications with subsequent cauterization with sulphate of copper, as recommended by Abadie and now by Giotti, were proposed and frequently performed by Ubandi 25 years ago. DANTONE.

TROUSSEAU (292) scraped a little off a conjunctiva affected with lupus, and experimented upon rabbits with it, introducing it in 2 cases into the corneal tissue and in 2 into the anterior chamber. In 3 of them he obtained positive results, as tubercles developed in the eye. Inoculation of the cornea is more difficult than inoculation in the anterior chamber; the iris in particular is very susceptible. MARCKWORT.

VOSSIUS (293) obtained good results in trachoma by excising the fornix. He recommends numerous sutures, however, in closing the wound, so as to prevent the growth of granulation-tissue.

294. FUCHS, E. Keratitis punctata superficialis. *Wiener klin. Wochenschr.*, 1889, No. 44.

295. GOTTSCHALK, J. Ulcus corneæ rodens. *Inaug. Diss.*, Bonn, 1889.

296. HIRSCHBERG, J. Keratitis in congenital syphilis. *Centralbl. f. prakt. Augenhk.*, 1889, p. 201.

297. LEPLAT. Sur la pathologie de la cornée. *Gaz. méd. de Liège*, 1889, p. 445.

298. Rosmini. Ancora nella cheratoplastica mediante inneste della cornea di coniglio. Quarta lettera al Prof. Gradenigo. *Boll d'ocul.*, vol. xi., 10–11, p. 73.

299. Secondi, G. Le iniezioni sottocongiuntivali di sublimato corrosivo per la cura delle alterazioni infettive della cornea. *Boll. d'ocul.*, vol. xi., 15–16, p. 115.

300. Walker, E. On the treatment of some of the grave diseases of the cornea. *Liverpool Med.-Chir. Jour.*, July, 1889.

301. de Wecker. Greffe cornéenne. *Soc. d'opht. de Paris*, May 7, 1889.

302. Williams, R. On three cases of conical cornea treated by the actual cautery. *Brit. Med. Jour.*, Feb. 23, 1889.

Stellwag, Adler, and v. Reuss have described keratitis punctata superficialis, which was first observed in Vienna. As the descriptions of these authors differed, however, **Fuchs** (294) has published his experience in 36 cases. The disease begins with the symptoms of an acute conjunctivitis with marked ciliary injection and profuse lachrymation, attended with photophobia and pain. After a few days small gray dots appear, sometimes in small, sometimes in large numbers, irregularly scattered over the cornea or principally near the centre. They lie in the superficial layers and raise the epithelium. The symptoms of irritation soon disappear, but the dots generally remain unchanged for months, and then slowly disappear. The disease, which Fuchs calls "keratitis punctata superficialis," most frequently attacks young people, and begins ordinarily with acute rhinitis and a cough. It closely resembles herpes corneæ febrilis, on account of the arrangement and location of the dots, but differs from it by the absence of vesicles in the face. Vesicles never occur upon the cornea, and very seldom loss of epithelium and ulcers.

Gottschalk (295) describes two cases of rodent corneal ulcer. He recommends treatment with the galvano-cautery.

According to **Hirschberg** (296), keratitis due to congenital syphilis always leaves blood-vessels in the cornea and generally also patches in the fundus.

Rosmini (298) transplanted a disc of rabbit's cornea into a leucomatous eye according to Hippel's method. For six days all went well. The disc became attached and remained clear; the patient declared after the removal of the bandage that she had seen the face and even the eye-glass of the operator. She did not remain quiet however after this, and disregarded the rules observed during the first days. In consequence the disc lost its transparency and came away after twenty-four hours. The eye was destroyed by panophthalmitis. DANTONE.

SECONDI (299) reports on the excellent results obtained at the Turin clinic with subconjunctival injections of sublimate. Solutions of 1:2,000, 1:5,000, and 1:10,000 were used, and the good effect sometimes was apparent within twenty-four hours, especially in purulent keratitis. The same method was also tried in endocular diseases with encouraging result, especially in syphilitic affections, and the author thinks the method has a great future.

DANTONE.

WALKER (300) recommends peritomy in severe corneal ulcers. His results were good. WERNER.

DE WECKER (301) performed transplantation of a rabbit's cornea according to Hippel's method, and then covered the eyeball with a glass shell which had previously been accurately fitted. The transplanted disc took hold, and through it could be seen what had been left of the human cornea, which unfortunately consisted of cicatricial tissue with adherent remnants of iris, so that no improvement of vision was obtained. De Wecker particularly calls attention to the good healing under the glass shell.

303. HERN, J. Notes of a case of sclerotitis apparently of dental origin. *Brit. Med. Jour.*, 1889, No. 1500, p. 698.

304. RAMPOLDI. Brevi considerazioni cliniche intorno a un caso di sclero-coroidite anteriore. *Ann. di ottalm.*, vol. xvii., 6, p. 544.

305. WALLSTABE, C. Contribution to the treatment of partial scleral staphyloma with the galvano-cautery. *Inaug. Diss.*, Greifswald, 1889.

HERN'S (303) patient, an otherwise healthy woman, æt. thirty-eight, experienced suddenly paroxysmal pain over the left infra-orbital region and towards the left eye. A patch of sclerotitis with corneal haze was observed at the lower, outer portion of the eye, which did not get well until after the removal of a carious upper bicuspid on the left side. WERNER.

RAMPOLDI (304) discusses a case of sclero-choroiditis anterior with advanced glaucomatous symptoms in a girl aged fourteen, whose mother suffered with gout. As no other cause could be found for the affection of the eye, the author is inclined to seek it in an hereditary arthritic diathesis, of which he had seen an analogous example in a young man a few years previously.

DANTONE.

WALLSTABE (305) recommends the repeated application of the glowing platinum wire in incipient traumatic scleral staphyloma.

Sections XII.–XXI. Reviewed by DR. P. SILEX.

XII.—IRIS.

306. ANDREWS, J. A. Primary sarcoma of the iris. *N. Y. Med. Jour.*, June 1, 1889.

307. BJERRUM. Et tilfalde af let iridocyclitis med. spontan arteriepuls in Art. centr. ret. *Nord. ophth. Tidsskrift*, vol. ii., 1, p. 23, Copenhagen, 1889.

308. HIRSCHBERG, J. Tuberculous tumor of the iris. *Berlin. med. Gesellsch.*, *München. med. Wochenschr.*, 1889, No. 22; *Deutsche med. Wochenschr.*, No. 23; *Allg. med. Centralztg.*, No. 50.

309. MCKEOWN, DAVID. Blind for sixty years. Iridectomy. Recovery of sight and of perception of color. *Memorabilia*, No. 7, p. 417.

310. LEPLAT. De l'iridocyclite chez les vieillards. *Ann. de la Soc. méd.-chir. de Liège*, 1889.

311. NOVELLI. Nota di un caso di riduzione d'ernia dell' iride. *Boll. d'ocul.*, vol. xi., 7, p. 49.

312. PARISOTTI. Iniezione di calomelano nella cura della sifilide oculare. *Boll. d. Soc. Lancisiana*, 1889.

313. STEFFEN, W. Fibrosarcoma of the globe in a child one and three quarter years old. *Jahrb. f. Kinderhk.*, vol. xxx., 1–2, p. 110.

ANDREWS' (306) case was that of a woman of forty-seven, who had noticed a discoloration of the iris at the site of the tumor for five or six years. Periodical obscurations of vision and injection frequent since that time. The tumor was excised with a piece of the iris by Dr. C. R. Agnew. A week afterwards the eye was enucleated, a general infection being feared. The patient was alive at last accounts. In addition to this case Andrews gives a short account of all the cases of a similar kind that have been reported, twenty-five in number. Most of the tumors seem to have originated in the anterior portion of the iris. In several cases sight was preserved after the removal of the tumor. BURNETT.

Bjerrum (307) reports a case of slight iridocyclitis in both eyes, in which all the arteries in the left eye showed an undulating pulsation on the papilla ; it presented the appearance of the ordinary venous pulse. Schiötz.

Hirschberg (308) presented a child eighteen months with apex-catarrh, upon whose iris a reddish-yellow tumor with numerous small nodules had developed. As an injury and syphilis could be excluded the diagnosis of tuberculosis was made. On October 23d he reported to the Medical Society that the eyeball had been removed, and that the well-known anatomical structure of the tubercle had been found, but no tubercle-bacilli.

Leplat (310) discusses several cases of iridocyclitis in older people which he had observed. The symptoms were generally as follows : ciliary injection, deposit on Descemet's membrane, discoloration of the iris, and more or less numerous posterior synechiæ, much reduced V, and T about + 1. In such cases great care must be exercised in the use of atropine when T is increased. They take the most favorable course under the following treatment : eserine, and, if necessary, puncture combined with subcutaneous injections of pilocarpine. (The Reviewer obtained good results from repeated instillations of pilocarpine into the conjunctival sac). Leplat also takes occasion to recommend the instillations of iodide of potassium in ocular diseases in which it is not borne by the stomach. [After I had seen this method of treatment recommended by Schweigger (*cfr. Arch. f. Augenhk.*, vol. xvii., p. 141), I have also used it frequently.—Rev.]

Marckwort.

Novelli (311) describes the successful reposition of a small hernia of the iris, which had existed less than twenty-four hours, and was the result of the perforation of a peripheral corneal ulcer.

Dantone.

In four cases of severe syphilitic iritis Parisotti (312) tried the subcutaneous injections of calomel and obtained surprising results : in twenty-four to forty-eight hours all the acute symptoms had disappeared. The author made the injection, not like other Italian colleagues in repeated doses of 10 cgr. at the temple, but in a single dose of 30 cgr. at the nates of the patients. The remedy, however, failed to remove the residues of an old keratitis parenchymatosa. Dantone.

In Steffen's (313) patient the fibrosarcoma sprang from the ciliary body, produced metastasis in the orbit, broke through posteriorly, and enveloped the greater part of the optic nerve. Numerous hemorrhages were found in the tumor. There were no metastases in other organs. The disease had lasted six months. The case is also remarkable on account of numerous hemorrhages under the cranial bones and marked rhachitic changes in the latter.

XIII.—CHOROID.

314. CHIBRET. Du curage antiseptique de l'œil dans la panophtalmie. *Rev. génér. d'opht.*, 1889, No. 5.

315. DEMME. Contributions to the knowledge of tuberculosis in childhood. Twenty-fifth med. report on the Jenner children's hospital at Berne, 1888.

316. GAYET. Sur un cas d'adénome de la choroide. (With illustrations.) *Arch. d'opht.*, vol. ix., No. 3, p. 205.

317. HIRSCHBERG, J. Incipient panophthalmitis with cystoid scar cured. *Centralbl. f. prakt. Augenhk.*, 1889, vol. xiii., p. 267.

318. TAILOR. Sulla tuberculosi cronica del globo oculare. *Ann. di ottalm.*, vol. xviii., 1–2, p. 76.

CHIBRET (314) describes a new operation which he performs in panophthalmitis. As soon as the diagnosis has been made, he operates as quickly as possible in the following manner: with Graefe's knife a large corneal flap is made downward, and the iris, pupillary exudations, and anterior capsule of the lens are extensively torn with the point of the knife, whereupon the lens escapes with the pus. Lukewarm antiseptic fluid (Hg_2OCy 1:1000) is then injected in various directions into the globe with an Anel syringe with a large canula, so that the suppurating part may be separated from the healthy, the latter remaining behind and being preserved, as Chibret maintains. The fluid last injected remains in the globe. The patients treated in this manner left the clinic three to six days after the operation. Chibret operated on five of them according to this method. In two of the cases Chibret estimated the stump which formed at two thirds of the size of the normal eye, in one at one half, of the other two he says nothing. (For cosmetic purposes such stumps are almost worthless, and they probably shrink in the course of time. An artificial eye, properly fitted, moves well without a stump; the latter is frequently irritated by the shell, while after an enucleation this is not possible. Whether the stump after Chibret's operation can give rise to sympathetic ophthalmia the future must show, as he himself correctly remarks. The patients however are to be pitied who must pay for this experience with the loss of the other eye.—Rev. MARCKWORT.

DEMME (247) reports on eighty-seven cases of miliary tuberculosis in children, in forty-nine of which it could be traced to primary tuberculous affection of the viscera, and in thirty-eight to a primary peripheric location. In nineteen he found tubercles in the choroid. In a blacksmith, thirty years old, without any hereditary taint, who four years previously had had attacks of fever in Cheyenne without particularly affecting his health, GAYET (316) observed a tumor of the choroid, which had been flattened to the shape of a lentil and touched the optic nerve with the outer side. The tumor presented the structure of tubular glands ; on cross-section it appeared marbled and pitted. The tubules, which seemed to have no membrana propria, were lined with cylinder-epithelium and contained granular cells with one or more nuclei. No inflammatory reaction was observable in the neighborhood of the tubules, which seemed to follow the course of the obliterated blood-vessels. The eye had become blind from detachment of the retina, and was very painful. This condition led to a careful general examination, which revealed the existence of carcinoma of the stomach. The cachectic patient soon died. Besides the carcinoma of the stomach several nodules were found in the liver. The author considers the choroidal tumor as secondary, an opinion which is supported by the similarity of its structure to that of the glands of the stomach. He thinks that choroidal tumors must be considered secondary growths more frequently than formerly. V. MITTELSTÄDT.

HIRSCHBERG (317) recommends the removal of the masses containing the microbes of suppuration by extracting them if movable, and burning them off when they are firmly united with the membranes of the eye.

TAILOR (318) describes two cases of tuberculosis of the eyeball, which was observed in girls aged seven and twenty-two respectively. The first was especially interesting, because the diagnosis of tuberculous neoplasm to the exclusion of glioma and sarcoma could only be made with the microscope, and because the tumor had partly perforated the sclera, partly had distended it considerably, and was covered with it as with a fibrous capsule.

DANTONE.

XIV.—GLAUCOMA.

319. DÜRR and SCHLEGDENDAL. Five cases of hydrophthalmus congenitus. *Graefe's Arch.*, vol. xxxv., 2, p. 89.

320. HIRSCHBERG, J. Almost absolute glaucoma in gummous iritis cured by iridectomy. *Centralbl. f. prakt. Augenhk.*, vol. xiii., p. 266.

321. HUTCHINSON, J. Introduction to a discussion on the treatment of glaucoma. *Ann. Meeting Brit. Med. Assoc.*, Aug., 1889.

322. KRÜCKOW, A. Remarks on glaucoma based on the statistics of 1,430 cases. *Wjestnik Ophth.*, July–Oct., 1889.

323. LAQUEUR, L. On glaucoma in young persons. *Arch. f. Augenhk.*, vol. xxi., 1, p. 91.

324. MITTERMAIER. On the occurrence of glaucoma in cataractous eyes. *Inaug. Diss.*, Heidelberg, 1889.

325. PRIESTLEY-SMITH. Erasmus Wilson lectures on the pathology of glaucoma. (Forty-eight illustrations.) *Brit. Med. Jour.*, Apr., pp. 757, 817, 880, 929.

DÜRR and SCHLEGDENDAL (319) present a very detailed description of the anatomical condition of five hydrophthalmic eyeballs, which showed great differences according to the complications. In one well-marked case they found partly old, partly fresh traces of inflammation at the sclero-corneal junction, also at the insertion of the iris, and signs of periphlebitis on the choroid, absence of Schlemm's canal to a great extent, obstruction of the anterior ciliary veins, almost total absence of the circular fibres of the ciliary muscle, atrophy of the iris, choroid, and retina, deep glaucomatous excavation and loosening of the dural sheath of the optic nerve. They also give very acccurate measurements of the insertions of the muscles, their width, and of the position of the varicose veins. The superior oblique muscle shows the peculiarity of a broad, transverse insertion, causing a depression. This insertion resembles that found by Stilling in many myopic eyes. The veins had a suprachoroidal sinus, which is supposed to be due to the peculiar form of attachment of the muscles and the compression of the veins at their exit depending upon it. No signs of inflammation were found in the choroid, as assumed by Birnbacher and Czermak as the cause of the dilatation of the veins. Finally a compilation of twenty cases of hydrophthalmus is given, in which the changes found in the different parts are discussed. According to the authors hydrophthalmus is a secondary glaucoma, the beginning of which must be sought in an intra-uterine inflammation of the choroid. In extra-uterine life venous stasis follows, the cause of which must be sought in the muscular pressure on the veins. In a patient suffering with gummous iritis of the right eye, with marked increase of tension, HIRSCHBERG (320) performed an iridectomy, whereupon the tension again became normal. As to the result of this discussion, which was introduced by JONATHAN HUTCHINSON (321), it was agreed on all hands that iridectomy was the most successful treatment for acute glaucoma. As to chronic glaucoma, opinions were of course divided, but the majority approved of iridectomy, and considered it superior to sclerotomy. Little could not corroborate the statement that iridectomy tends to further contract the field of vision, and advocated the operation in the early or premonitory stage. Frost had had an eye lost by intraocular hemorrhage after an early operation, and hence does not interfere until the field begins to shrink. Mules recommends a very small iridectomy upwards, to be followed by a very large sclerotomy three days later. Berry alluded to the scarcity of glaucoma in Scotland, a fact which Hill Griffith thought might be due to the relative infrequency of gout in that country. Swan Burnett observed that the black race, free as it was from trachoma and other eye diseases, had its full share of chronic glaucoma. Pilocarpine, on the whole, was considered preferable to eserine. WERNER.

KRÜCKOW (322) disproves the opinion of Bonwetsch that the Slavs are more disposed to glaucoma than other European nations. Based on his statistics, Krückow also opposes the opinion that hypermetropia predisposes to glaucoma. There were among his glaucoma patients 28 % with emmetropia, 26.78 % with myopia, and 43.18 % with hypermetropia. The preponderance of glaucoma patients with hypermetropia he ascribes to the greater frequency of hypermetropia in itself. The greatest number of glaucoma cases is observed between the ages of fifty and seventy-five. Sex made no material difference. The right and left eye were affected with equal frequency. The time elapsing between the affection of the first and second eye varied up to fifteen years. In one case the other eye was still healthy after seventeen years.

HIRSCHMANN.

MITTERMAIER (324) has compiled twenty-one cases, among them five from the Heidelberg clinic, in which glaucoma was observed in cataractous eyes, and comes to the following conclusions: In aging eyes primary glaucoma often develops with cataract. A connection between the two has not been proven. Aphakia does not protect against glaucoma, not even when an iridectomy is performed at the time, or as a preliminary operation. When the operation was irregular, or complications set in in the course of healing, the glaucoma is but secondary. If this is not the case, primary glaucoma developing later resembles those cases in which an iridectomy produced only a temporary improvement, and had to be repeated.

PRIESTLEY-SMITH (325) discusses at great length, in three lectures, based on original investigations, the whole question of the pathology of glaucoma. In Lecture I. the author draws attention to the fact that increased tension, which is the essence of the disease, is frequently intermittent, and may even be absent for long periods of time. To understand the morbid process, we must study first the normal intraocular pressure and the processes by which it is maintained; secondly, the causes which lead to an excess of pressure; and thirdly, the consequences of such excess.

Under the first head the principles of tonometry, and the secretion and escape of aqueous fluid, are treated of and summarized as follows: The fluids which nourish the vitreous body and lens and fill the aqueous chamber are secreted by the ciliary portion of the uveal tract. The larger part of the secretion passes directly into the aqueous chamber, forwards through the pupil, and out at the filtration-angle. A very much smaller portion passes backwards through the vitreous body, and escapes at the papilla. The hyaloid membrane and zonula which separate the two chambers are readily permeable by the vitreous fluid. The pressure which the fluid exerts against the walls of the chambers is equal to about 25 *mm* of mercury, and is the same, or nearly so, in both chambers. Lecture II. first treats of the physical and physiological experiments bearing on increased tension; a modification of Birnbacher and Czermak's apparatus is described and figured. Of the causes which may give rise to glaucoma—viz.: hypersecretion of the ciliary processes, obstruction at the filtration-angle, and serosity of the fluids, the second one is in the author's opinion the most important. Among seventy or more eyes examined, representing various forms of glaucoma, only three failed to show compression of the filtration-angle. When this sign is present without glaucoma, the fluid finds an abnormal outlet, *e.g.*, through a diseased cornea, or else the ciliary processes are atrophied or damaged, and secretion is arrested. On the other hand, serosity of the fluids of the eye may give rise to increased tension without obstruction of the filtration-angle, as in serous iritis. The closure of the filtration-angle is a cause of the high pressure, and not a consequence of the latter, in cases of annular posterior synechiæ, perforating wounds and ulcers of the cornea, dislocations and injuries of the lens, and in intraocular tumors which produce secondary glaucoma. A case of glaucoma after cataract-extraction is described, in which puncture of the membrane in the coloboma resulted in a gush of fluid from behind, and the eye recovered its normal tension. Numerous illustrations of eyes which suffered from secondary glaucoma accompany the paper.

Lecture III. The author is thoroughly convinced, after examining twenty-five specimens of primary glaucoma, that in all cases the ciliary processes press against the base of the iris, and thereby close the filtration-angle. Birnbacher and Czermak's observations (*v. Graefe's Arch.*, vol. xxxii., p. 1) are unreliable, both as regards material and method. As regards predisposing causes, there is a parallelism between the increasing liability to glaucoma and the increasing size of the lens which accords well with the supposition that a diminished circumlental space predisposes to the disease. The smallness of the cornea in glaucomatous eyes is another point in favor of this view. Having measured a large number of healthy and glaucomatous eyes, twenty-eight persons were found to have small corneas, and among these twenty-eight twenty had glaucoma. Microphthalmic eyes with healthy lenses are also prone to glaucoma. Excessive size or prominence of the ciliary region may also tend to diminish the circumlental space, *e.g.*, Fuchs' case (*v. Graefe's Arch.*, vol. xxx., 3, p. 123). Phlebitis of the vertex veins has been described as a cause, but the author has found these changes in the veins in cases of secondary glaucoma where they were certainly not the initial causes of the disease. Treatment by eserine is not only useless but injurious if it does not contract the pupil, as it increases hyperæmia. It is better to use the weakest solution which will contract the pupil, and it should be combined with cocaine. Werner.

XV.—SYMPATHETIC OPHTHALMIA.

326. **Abadie.** Des diverses formes cliniques de l'ophtalmie sympathique. *Soc. franç. d'opht.*, Aug. 9, 1889.

Abadie (326) distinguishes two kinds of sympathetic ophthalmia: (1) The infectious, the existence of which has been proven by Deutschmann beyond doubt, and which takes its way through the optic nerve. In this form the optic nerve and its surroundings are first affected; the vitreous next becomes cloudy, and finally the uveal tract becomes diseased, especially the iris. In this form the focus of infection in the primarily affected eye must be treated with the galvano-cautery, and eventually the anterior chamber must be emptied. Mercurial treatment is decidedly indicated. If no improvement thereupon takes place in the other eye, enucleation must be resorted to. (2) The reflex form, which is transmitted through the ciliary nerves. For the treatment of this form, the existence of which has not been positively proven, Abadie recommends sclerotomy, which divides the ciliary nerves between the wound and the nerve-centre. According to Wecker, surgical procedures in traumatic cataract must be especially feared on account of sympathetic ophthalmia. Panas considers enucleation very good prophylactic treatment. After the disease has developed, however, mercurial treatment gives the best results. Dransart also did not observe any improvement from enucleation after sympathetic ophthalmia had once developed; he therefore removes, as a prophylactic measure, every eye irretrievably ruined by an injury. Reymond obtained good results in sympathetic ophthalmia with subconjunctival injections of sublimate (1:2,000 to 500). Nuël found in eyes which had caused the disease only staphylococcus albus and aureus.

Marckwort.

XVI.—LENS.

327. **Abadie.** De certaines complications qui surviennent quelques jours après l'opération de la cataracte. Pathogénie. Traitement. *Ann. d'ocul.*, vol ci., p. 179.

328. **Ayres, S. C.** Extraction of cataract without iridectomy, or simple extraction. *Amer. Jour. Ophth.*, May, 1889.

329. **Baudon.** Note sur les résultats obtenus dans cent quarante opérations de cataracte. Short report on 140 cataract operations (partly extractions, partly divisions), with 13 failures. *Rec. d'opht.*, No. 7, p. 395.

330. Blanch. Quelle est la meilleure méthode d'extraction de la cataracte ? *Rec. d'opht.*, 1889, No. 8, p. 460.

331. Critchett. Address delivered at the opening of the Section of Ophthalmology. *Brit. Med. Jour.*, Aug. 24, 1889, p. 414.

332. Despagnet. De l'extraction du cristallin luxé. *Soc. d'opht. de Paris*, May 7, 1889.

333. Fage, A. Contribution à l'étude des hemorrhagies intra-oculaires consécutives à l'extraction de la cataracte. Examen histologique. *Arch. d'opht.*, 1889, vol. ix., No. 4, p. 351.

334. Galezowski. Quelques mots sur l'extraction de la cataracte et sur l'application dans quelques cas d'iridectomie. *Soc. d'opht. de Paris*, July 2, 1889.

335. Graefe, A. Continued report on the results of cataract extractions under antiseptic precautions. *v. Graefe's Arch.*, vol. xxv., 3, p. 249.

336. Gunn. Note on the action of the aqueous on lenticular substance. *Ophth. Rev.*, vol. viii., p. 235.

337. Hirschberg, J. On cataract operations and diabetic senile cataract. *Deutsche Med. Wochenschr.*, 1889, No. 37.

338. Kazaurow, J. The question of operating for cataract by aspiration. *Wjestnik Ophth.*, July–October, 1889.

339. Kolinski, J. The action of naphthaline upon the eye and the so-called naphthaline cataract. *v. Graefe's Arch.*, vol. xxxv., 2, p. 29.

340. Lawrentjew, L. K. Report on a third hundred of cataract extractions. *Wjestnik Ophth.*, May–June, 1889.

341. Martin, E. La suture de la cornée dans l' opération de la cataracte. *Rec. d'opht.*, 1889, No. 5, p. 265.

342. Nicolini. Sulla maturazione artificiale della cataratta. *Boll. d'ocul.*, vol. xi., 15–16, p. 105.

343. Panas. L' action thérapeutique de l'antipyrine dans la glycosurie. *Arch. d'opht.*, vol. ix., No. 3, p. 265.

344. Parisotti. Sulla maturazione artificiale della cataratta. *Rif. Med.*, Jan., 1889.

345. Risley, T. D. Incipient cataract; its etiology, treatment, and prognosis. *University Med. Magazine*, Mar., 1889.

346. Schies. Report on 1,100 cataract operations. *Schweizer Correspondenzbl.*, 1889, No. 15, p. 461.

347. Steffan, Ph. Further experiences and studies on cataract extraction, 1882–1888. Antiseptics and technique. *v. Graefe's Arch. f. Ophth.*, vol. xxxv., 2, p. 171.

348. Suarez de Mendoza. Suture de la cornée dans l'opération de la cataracte. *Rec. d'opht.*, 1889, No. 9, p. 523, and *Soc. franç. d'opht.*, Aug. 8.

349. Teale, A. Discussion on the treatment of immature cataract. *Brit. Med. Jour.*, Sept. 28, 1889, p. 694.

350. Thorn, H. On suppuration after cataract extraction. *Inaug. Diss.*, Greifswald, 1889.

351. de Wecker. L'avenir de l'extraction de la cataracte. *Ann. d'ocul.*, vol. ci., p. 221.

352. de Wecker. Remaniement du procédé classique de l'extraction simple. *Soc. franç. d'opht.*, Aug. 8, 1889.

353. Wicherkiewicz, Bol. The most suitable method of opening the capsule for cataract extraction. *Klin. Monatsbl. f. Augenhk.*, vol. xxvii., p. 169.

354. Wicherkiewicz. Instruments pour l'opération de la cataracte. *Soc. franç. d'opht.*, Aug. 8, 1889.

Abadie (327) believes that the symptoms of inflammation suddenly appearing 5 or 6 days after a successful extraction are due to infection, the development of which was delayed for a while by the antiseptics used. He advises cauterizing the wound in such cases with the galvano-cautery. Marckwort.

In the 32 cases of simple extraction done by Ayres (328), there was one failure, due to injury inflicted by the patient himself. On the whole the author is favorably impressed with the operation. Burnett.

Blanch (330) extracts cataracts without irridectomy and opens the capsule with the Graefe knife according to Galezowski, immediately after the puncture. The corneal incision lies in the limbus a little above the horizontal diameter, and the height of the flap is about 2—2½ *mm* from the edge. The knife is the only instrument introduced into the eye. On the basis of his own experience he is strongly opposed to washing out the anterior chamber. Marckwort.

Despagnet (332) describes the successful extraction of a dislocated lens with a broad spatula. Trousseau extracted 13 dislocated lenses with Taylor's loop; he makes a small corneal section. Gillet de Grandmont also recommends the extraction of dislocated lenses, but with a large corneal section. (Any one who has extracted according to Pagenstecher hypermature or otherwise suitable cataracts within the capsule will be surprised at still seeing so many oculists hesitate about extracting a dislocated lens, provided it is not dislocated to such an extent that it cannot be seized with the spoon, or when severe complications exist. Rev.) MARCKWORT.

FAGE (333) reports on a case of intraocular hemorrhage after extraction with iridectomy, observed at Badal's clinic. The cataract was not complicated, and there was no myopia or increase of tension. The patient, a man aged fifty-nine, was free from any diathesis, no atheroma or heart-disease (whether a bleeder is not mentioned. Rev.), had only slight bronchitis, and cough. He had scarcely gone to bed, whither he had slowly walked, as is customary in Badal's clinic, when he felt the hemorrhage, which lasted several days, in spite of cold applications and a compressive bandage. The eye was enucleated on the twelfth day, after panophthalmitis had developed, when a large bloodclot was found between the choroid and retina. The hemorrhage, therefore, probably came from the choroid, the arteries, of which, however, did not show any fatty or atheromatous changes. The author was unable to discover the cause of the hemorrhage.

v. MITTELSTÄDT.

GALEZOWSKI (334) again discusses his method of extracting cataracts, and once more recommends his gelatine tablets for covering the wound. Iridectomy he advises in the following cases: (1) Where the iris falls before the knife; (2) in synechiæ; (3) when the corneal wound is too small; (4) in capsular cataract; (5) in subluxation of the lens; (6) in constitutional cataract (glycosuria, albuminuria); (7) in syphilis. Parent adds to this in the discussion, cases of increased tension. The discussion had reference mainly to the time when the first bandage should be changed. Galezowski changes it for the first time on the sixth day; Parinaud after 24 hours, on account of the incarceration so frequent in extraction without iridectomy. In such a case he performs iridectomy a day after the extraction, which at a later period is no longer possible. Despagnet does not change the bandage for a week, in order to prevent hernias of the iris due to re-opening of the wound. During the first 24 hours he irrigates the bandage with a saturated solution of boracic acid, thus making it firm by crystallization within it. Valude does not change the bandage for 3 or 4 days. Gutierrez Tome recommends the glass shells which adapt themselves to the eye; Despagnet is opposed to them, as, according to his experience, simple closing of the lids is sufficient to break them.

Marckwort.

Graefe (335) lost only 0.93 per cent. from suppuration among 1,074 extractions in which he used the antiseptic precautions described in *Graefe's Arch.*, vol. xxx., 4, p. 217. From this he concludes that we must expect the best results, not from technique, as Steffan assumes (*v. Graefe's Arch.*, vol. xxxv., 2), but from asepsis and antisepsis. The inundations of the eye with a warm solution of sublimate before, during, and after the operation were probably the cause, together with cocaine, of the marked and permanent corneal opacities in 4.7 per cent. of all cases.

Gunn (336) suggests that the solution of cataract is due to the solubility of globuline in weak solutions of sodium chloride (aqueous humor). Werner.

Hirschberg (337) explains why extraction without is to be preferred to extraction with iridectomy, and gives a detailed description of his method, the preparation of the instruments, etc. He reports on sixty-two cases, among them eight with diabetes.

Kazaurow (338) resorted to aspiration for removing soft masses of cataract in five cases. Three were cases of swelling lens-masses after division or injury, two were soft diabetic cataracts in the same individual. Bowman's aspirator was introduced through an opening which had been made with a small knife in the cornea and capsule, strict antiseptic precautions being observed. The results were satisfactory, in the diabetic cataracts even brilliant. After two days the eye was free from reaction. Kazaurow considers this method indicated in zonular cataract after division, in congenital soft cataract, in traumatic cataract, and diabetic cataract of young persons. Hirschmann.

Kolinski (339) ascribes the changes produced by naphthaline to alterations in the walls of the blood-vessels. The changes in the lens are always preceded by those in the retina, which are themselves secondary. For details see original.

Lawrentjew (340) performs sphincterectomy instead of iridectomy in extraction, is very well satisfied with it, and is opposed to simple extraction without iridectomy. He had no suppuration, but in three cases $V \frac{1}{\infty}$. Hirschmann.

MARTIN (341) is strongly opposed to the corneal suture recommended by Suarez de Mendoza. He considers it not only unnecessary but also dangerous. He is one of the few French oculists who still adhere to the combined extraction.

MARCKWORT.

NICOLINI (342) reports on ten cases of artificial ripening of cataract by trituration of the cortex after a previous iridectomy. It was attended with accidents in only two cases. In one there was considerable hemorrhage into the anterior chamber, in the other the capsule burst and part of the cortex escaped. The result of the subsequent extraction was very satisfactory in eight cases ; an immediate failure occurred in only one patient, who suffered with albuminuria, and died of the kidney-disease a few weeks later. The extraction was done successfully in the eye in which the capsule burst ; the patient saw when discharged, but soon lost his sight from glaucoma. DANTONE.

PANAS (343) recommends antipyrine in doses of 3 *grms* in diabetes. He extracted two diabetic cataracts with good result. In the one the sugar could not be made to disappear in any way. After giving, however, 3 *grms* of antipyrine, and enforcing strict diet, he saw the sugar, which had amounted to 30 *grms* daily, completely disappear after six days. After the operation 2 *grms* of antipyrine were given daily, and the sugar did not reappear until 150 *grms* of bread were allowed, when it amounted to 4 *grms* a day. In the second case the effect was also very satisfactory. v. MITTELSTÄDT.

PARISOTTI (344) resorted to the artificial ripening of cataract in two cases by trituration of the cortex after simple puncture of the anterior chamber without iridectomy, and was well satisfied with the result. He calls special attention to the absence of any reaction on the part of the iris, which of course had been rubbed also. DANTONE.

RISLEY (345) thinks that while cataract is a disease of advanced life, it does not depend entirely on senile change, but is due to pathological states, involving the nutrition of the eye itself. The reports of several cases are given in support of this view.

BURNETT.

After an historical review of cataract extraction, SCHIES (346) describes the method practised at the Basel clinic. He favors a flap-section at the sclero-corneal margin under strict antiseptic precautions. In the tenth and eleventh hundred he had no losses whatever. Danger of the prolapse of iris is the obstacle to extraction without irridectomy.

Steffan (347) shows that in spite of all antiseptic precautions it is impossible absolutely to disinfect the conjunctival sac, and that the antiseptic bandage is therefore useless, as it cannot keep the corneal wound aseptic. In spite of antiseptic measures he had a loss of 6.6 per cent. from infection, and of 6.7 per cent. when only aseptic precautions were resorted to. Steffan makes a peripheral flat, downward flap, combined with iridectomy, and opens the capsule with a horizontal incision. Since July, 1888, he always performs a preliminary iridectomy.

Suarez de Mendoza (348) again discusses suture of the cornea after extraction (*cfr.* these Archives, vol. xviii, p. 351, No. 534). Of the numerous details, the manner in which the suture is applied is particularly interesting : at the upper end of the vertical diameter of the cornea he makes an incision in the cornea with a Graefe knife, 5–6 *mm* long, parallel to the margin, beginning on the outer side and extending inward, through about two thirds of the thickness of the cornea, and therefore not opening the anterior chamber. Into this wound he introduces a loop of silver wire, which he holds in a fixation-forceps. With the aid of a curved needle, 15 *mm* long, he passes a silk thread through the upper edge of the wound, through the loop, and then through the lower edge. The needle does not enter the anterior chamber. The points of entrance and exit are about 1 *mm* from the edge of the wound. By withdrawing the loop from the incision he also pulls out the thread in form of a loop. After cutting the silver wire the loop of thread is pushed aside, and the cataract removed in the usual manner, though the precaution must be observed of making the cataract incision coincide exactly with the one previously made. After the operation the wound is closed with two knots. If necessary one suture more is introduced on each side, though they are generally omitted. In this manner Suarez de Mendoza closed the wound after extraction in four rabbits, one dog, and fifteen human beings. The threads remained in position 5–10 days. Gayet thinks the corneal suture is principally adapted for cases with increased or diminished tension. Gillet de Grandmont and Vacher also favor the corneal suture.

Marckwort.

Teale (349) has found the old flap operation more satisfactory than Graefe's operation with iridectomy. Since 1873 he performs what he calls the "shallow flap." A Sichel knife is entered in the horizontal meridian of the cornea, and there abruptly turned forwards, producing a shallow, angular flap. He performs preliminary iridectomy in feeble patients, and when the first eye goes wrong.—Snell read a paper on the extraction of cataract with and without iridectomy: an analysis of 300 operations. The author performs a corneal incision downward without the assistance of speculum or fixation forceps. Iridectomy was performed in only one third of the operations. Eserine was used in only a few cases. Vitreous was lost in six per cent., and prolapse of iris occurred in one per cent. of the cases. Of twenty failures, eleven occurred in the iridectomy series, six in the seris without, although the former were only about a third of the latter in number.—Hewetson adopts Teale's method, and is very careful as to antisepsis. He clips the eyelashes before operating. Prolapse of the iris was very rare.—Berry described the antiseptic method he employed. The inoculation through instruments could be readily and completely avoided; it therefore remained to diminish the risk of inoculation from sources within the eye. As regards the cornea, this could be done by frequent washing of the wound during operation, making a clean cut through the cornea, without sawing movements, and guarding against bruising the edges of the incision. Septic iritis is more difficult to overcome, and a preliminary iridectomy is the best mode of avoiding it.—Little adhered to the iridectomy, and had obtained excellent results. Teale had not given his results, nor had Snell done so with sufficient detail.—Mules thought the safest method was preliminary iridectomy. He agreed with Meyer, of Paris, that there are patients who seem to carry the source of infection in themselves.—Frost's chief objection to the operation without iridectomy was the stretching and bruising of the iris; another difficulty was the clearing out of the cortex. Too much importance had been attached to the cosmetic effect. WERNER.

THORN (350) describes five cases of suppuration observed at the Greifswald clinic, and as the usual antiseptic precautions were observed, and the operations regularly performed, the suppuration must be attributed to diminished power of resistance of the tissue to the microbes, and to the impossibility of attaining strict antisepsis in operations on the eye.

DE WECKER (351) answers the paper of Alfred Graefe (*Arch. f. Ophth.*, vol. xxxiv., 3, p. 323). He is opposed to the exclusion of iridectomy as a principle, and performs it in one third of all cases. Among 287 extractions there were 92 with iridectomy.

MARCKWORT.

DE WECKER (352) frequently found in division of congenital cataracts a tough capsule and a hard nucleus. In children with congenital cataract (not zonular) he therefore now makes an incision with a lance-shaped knife at the upper corneal margin, and seizes the anterior capsule with the capsule-forceps. If he finds decided resistance, he enlarges the wound, makes an iridectomy, and extracts as usual. In congenital cataracts he thinks division should no longer be practised.—Panas agrees with him.—Landolt and Terson oppose Wecker's opinion, as prolapse of iris readily occurs in children, and because an enlarged is always an irregular wound. MARCKWORT.

As extraction within the capsule is practicable in only a few cases, WICHERKIEWICZ (353) recommends at least the removal of as large a piece as possible. He obtained very good results with a capsule-forceps designed by himself, and made by Haertel in Breslau, and constructed after the principle of Noyes' scisssors.

WICHERKIEWICZ (354) again shows his undine for washing out the anterior chamber, and his capsule-forceps.—de Wecker remarks that he used such a forceps as early as 1871.—Galezowski regards the opening of the capsule with forceps as dangerous.—Martin recommends iridectomy and the use of the capsule-forceps. (As the ordinary capsule-forceps easily also grasps the iris when no iridectomy has been made, Wecker has given it a peculiar form, so that iridectomy is no longer necessary when this instrument is used.) MARCKWORT.

XVII.—VITREOUS.

355. RANDALL, A. On the nasal reflex crescent of Dr. L. Weiss. *Klin. Monatsbl. f. Augenhk.*, vol. xxvii., May.

356. VALK, F. Fatty degeneration of the vitreous body. *N. Y. Med. Rec.*, May 25, 1889.

RANDALL (355) saw the phenomenon in only six per cent. of myopic eyes. In two cases he could trace it, not to detachment of the vitreous, but to marked elevation of the retinal fibres at the medial half of the papilla.

VALK (356) reports a case in which there were seen in the vitreous of an eye, which seemed otherwise healthy, numerous globular, white, glistening spots, which he thinks may be due to fatty degeneration of that body. BURNETT.

XVIII.—RETINA AND FUNCTIONAL DISTURBANCES.

357. Becker, F. A case of amblyopia due to carbon bisulphide. *Centralbl. f. prakt. Augenhk.*, vol. xiii., p. 188.

358. Brunschwig. Diplopie monoculaire. *Rec. d'opht.*, 1889, p. 468.

359. Galezowski. Du traitement du décollement de la rétine. *Soc. d'opht. de Paris*, April 2, 1889.

360. Gelpke. A case of detachment of the retina treated according to Schöler with fatal result. *Centralbl. f. Augenhk.*, vol. xiii., p. 260.

361. Guaita. Sulla distinzione ottalmoscopica del pigmento retinico e coroideale e sulla patogenesi dell' emeralopia. *Ann. di ottalm.*, vol. xvii., 6, p. 501.

362. Hirschberg, J. Degeneration of the macula lutea in old people. *Centralbl. f. prakt. Augenhk.*, vol. xiii., p. 258.

363. Hirschberg, J. The variations in the width of the retinal blood-vessels. *Centralbl. f. prakt. Augenhk.*, vol. xiii., p. 203.

364. Hösslin. Contribution to the diagnostic measurement of the visual field in neuroses. *Münchener. med. Wochenschr.*, 1889, No. 6.

365. Ljubinski, A. Ophthalmia photo-electrica. *Wjestnik Ophth.*, May–June, 1889.

366. Novelli. Un caso di retinite albuminurica in puerperio. *Boll. d'ocul.*, vol. xi., 14, p. 97.

367. Nuel et Leplat. Troubles visuels dans l'anémie causée par le presence de vers intestinaux. *Ann. d'ocul.*, vol. ci.

368. Nuel et Leplat. Amblyopie due à l'intoxication par le sulfure de carbone. *Ann. d'ocul.*, vol. ci., p. 145.

369. Raehlmann, E. On a pulsating aneurism of the central retinal artery. *Klin. Monatsbl. f. Augenhk.*, vol. xxvii, p. 203.

370. Westhoff. Iridectomy in detachment of the retina. *Kliniek voor Ooglijders*, 1889, p. 8.

Becker's (357) patient is a man aged sixty-six, who was engaged in the manufacture of vulcanized rubber, and who came on account of a diminution of sight, which had developed after a feeling of general discomfort. V = fingers at 3 ft. Good sight was regained after the injuries had ceased to act upon him.

Fundus normal. There was a large central scotoma, but the periphery of the visual field was not affected.

BRUNSCHWIG (358) that in a case of monocular diplopia observed by himself hysteria was the cause. A girl aged eleven, who had had convergent strabismus of the right eye for six years, with vision in it of $\frac{1}{4}$, saw double when the left eye was covered: a stick of sealing-wax held vertically before the right eye was seen as two vertical sticks, held horizontally as two horizontal sticks above each other. The distance apart of the images remained the same, no matter what was the distance of the object from the eye. With both eyes open she saw three images for a moment, though the two of the right eye were immediately suppressed. The four wafers arranged in the usual manner were seen as four in the stereoscope, therefore without binocular fusion, but without double vision of the right eye. The visual field of both eyes was decidedly contracted, that of the right more than that of the left. MARCKWORT.

On the basis of three cases GALEZOWSKI (359) recommends subretinal injection of tincture of iodine in detachment of the retina, combined with a suture of the retina with catgut.

MARCKWORT.

GELPKE (360) injected under observance of strict antiseptic precautions three drops of tincture of iodine into the preretinal vitreous space in a robust man of sixty-six, who had had detachment of the retina of the left eye for a year. Purulent choroiditis ensued, followed by acute meningitis, of which the patient died six days after the operation. A detailed anatomical investigation will be given later.

GUAITA (361) presents a detailed account of the anatomical and ophthalmoscopic condition of the choroidal pigment and the pigment-epithelium of the retina, the connection of the latter with the visual purple and the development of hemeralopia, and finally describes the successive changes of the tissue in the development of retinitis pigmentosa. DANTONE.

HIRSCHBERG (362) gives an accurate description of the well-known ophthalmoscopic appearances. In old people there is a diminution of the acuteness of central sight with full extent of the visual field. Blindness does not ensue. The ophthalmoscope shows rose-colored spots, within them white dots with crystals, more recent grayish-blue patches behind the blood-vessels, and a blackish stippling of the periphery.

HIRSCHBERG (363) calls attention to the fact that the irregularities in the width of the blood-vessels, especially in retinitis albuminurica, can frequently be observed with the ophthalmoscope before the appearance of the characteristic stellate figure. He cites three cases in support of this assertion.

HÖSSLIN (364) found that the concentric contraction of the visual field is found not only in hysteria, but also in numerous functional neuroses (intestinal neuroses, epilepsy, neurasthenia, etc.). Sector-shaped defects of the visual field are not observed in these neuroses.

LJUBINSKI (365) saw a peculiar affection of the eyes, produced by gazing with unprotected eyes at the glowing carbon points of strong electric illuminating apparatus. Gazing at them less than a minute to three minutes was sufficient to produce the affection. The author saw about 30 such cases, generally in young naval officers, who were experimenting with various illuminating apparatus. In some of them the affection was observed several times (in one nine times). It seems that a relapse easily occurs. As the exact nature of the affection is not known as yet, the author proposes to call it temporarily "ophthalmia photo-electrica." After a strong electric light has been gazed at, after-images prevail for some time; white objects seem yellowish for a while. The eyes then become quiet for the whole day (12–14 hours). Sometimes the patient can still read at night without trouble. The symptoms of the disease always appear during sleep (even during an afternoon nap). The patients awake with severe pain in the eyes, as though they were full of sand and the lids filled with quicksilver. There is profuse lachrymation, and even the slightest amount of light causes great pain. The lids and conjunctiva are swollen, the latter is much injected, marked pericorneal injection. To move the eyes is painful. Touch is not resented. The tension is not increased. The attack lasts one and one-half to three hours, sometimes longer. The patient then falls asleep and awakes the following morning with almost well eyes: no redness, no pain, no discharge, at most a tired feeling, like that after long continued reading. In some patients the symptoms reappear the following night, or even two nights, but in a much less degree. The day after the attack there is marked hyperæmia of the retina and papilla, sometimes also venous pulse. In one case, after four attacks, Ljubinski saw at the yellow spot small round yellow dots, but no corresponding scotomas. The author did not succeed in determining whether light, heat, or chemical influences played the most important part in the etiology of this affection. He looks for the seat of it both in the retina and in the cornea. He found that dark-gray glasses afforded the best protection against strong light; blue glasses are also useful. Glasses of other colors seem to be useless. Treatment consists in cold applications, instillations of cocaine and internal exhibition of chloral. HIRSCHMANN.

NOVELLI'S (366) patient, a woman aged 27, became totally blind in childbed in both eyes. Extensive retinitis albuminurica was found. After energetic general treatment, sight gradually returned to the normal, though it required more than half a year. No traces of the disease remained except a dirty-white discoloration of the papilla. DANTONE.

NUEL and LEPLAT (367) found among 81 patients suffering with amchylostomum duodenale 6 with diseases of the eye: they were neuroretinitis and retinal hemorrhages, with hemorrhages and nystagmus. MARCKWORT.

NUEL and LEPLAT (368) saw in two working-women amblyopia due to bisulphide of carbon. The characteristic feature was the high degree of amblyopia (V figures at 3 ft.) with normal visual field and color-perception. The central scotoma was very small; at the distance of the radius of the perimeter it had a diameter of only ½ *cm.* The ophthalmoscopic changes were very slight. The edge of the papilla appeared a little indistinct. The patients completely recovered. MARCKWORT.

RAEHLMANN (369) has compiled the few communications on aneurisms of the retinal vessels and describes three cases observed by himself, one of which, that of a farm-hand aged 32, is of interest, inasmuch as the end- and periarteritic changes generally visible with the ophthalmoscope were not seen. From the position of the spindle-shaped aneurism situated upon the papilla and its relation to the distribution of the blood-vessels—rectangular forking of the central arteries upward and downward and no further division until beyond the edge of the disc—he concludes that the dilatation was due to mechanical action, and assumes that the anterior wall was unable to withstand the pressure on account of the rectangular division of the blood-vessels.

WESTHOFF (370) had treated a detachment of the retina in a man aged 35 with rest and injections of pilocarpine, without result. An iridectomy restored sight and the defect in the visual field disappeared. WESTHOFF.

XIX.—OPTIC NERVE.

371. Calderon. Double névrite optique; kyste hydatique intra-cérébral. Its existence was shown anatomically. *Soc. Franç. d'opht.*, Aug. 9, 1889.

372. Darier. De la suspension dans les atrophies des nerfs optiques. *Soc. d'opht. de Paris*, March 12, 1889.

373. Kalt. Double névrite optique avec myelite diffuse aiguë. *Soc. Franç. d'opht.*, Aug. 12, 1889.

374. Kny, E. A case of isolated tumor of the pituitary gland. *Neurol. Centralbl.*, 1889, No. 10.

375. Meyer, P. On a case of cerebral tumor. *Charité Annalen*, vol. xiv., p. 623.

376. Oppenheim. A case of cerebral tumor. *Neurol. Centralbl.*, 1889, No. 10.

377. Styx. A case of otitis muco-purulenta with endocranial complications. *Zeitschr. f. Ohrenhk.*, vol. xix., p. 244.

378. Sym, G. Tumors of the optic nerve. *Brit. Med. Jour.*, Sept. 28, 1889, p. 717.

379. Valude. Restitution ad integrum d'une lésion optique d'origine cerebrale. *Soc. d'opht. de Paris*, March 12, 1889.

Darier (372) advises suspension according to Charcot in tabic atrophy of the optic nerve. The improvement he obtained was probably more subjective than objective, although V is said to have risen from $\frac{1}{4}$ to $\frac{1}{2}$ and from $\frac{1}{6}$ to $\frac{1}{3}$. According to Wecker, Landolt, Gillet de Grandmont, Parninaud, and Abadie, suspension is useless in tabic atrophy of the optic nerve. Marckwort.

Kny's (374) patient first came under treatment in 1882, and became blind on February 1, 1889, on account of binocular optic neuritis. Divergent strabismus, nystagmus, dementia, and epileptiform attacks afterwards developed. The autopsy revealed a round-celled sarcoma of the pituitary gland, and firm adhesions around the optic nerves, which, like the tractus, looked like diaphanous bands. No positive symptoms indicating the seat of the lesion existed.

Meyer's (375) patient is a boy aged five, who became ill under the symptoms of cerebral pressure, had choked disc, and paralysis of the facial and abducent nerves of the right side. A peculiar chirping sound could be heard all over the skull. The autopsy revealed a very vascular glioma, which sprang from the floor of the fourth ventricle.

Oppenheim (376) found choked disc in both eyes of his patient. There were symptoms indicating a lesion of the left motor zone. Here a large round-celled sarcoma was found at the autopsy.

At the beginning of the third week vomiting, pain at the back of the neck, paralysis of the abducent, and moderate neuritis developed in Styx's (377) patient, both of whose eyes were affected. There was also some fever. The inflammation is supposed to have spread to the cavernous sinus, thus explaining the ocular symptoms. The paralysis had disappeared when the patient was discharged, but there was still hyperæmia of the disc.

In Sym's (378) case, a girl aged three, exophthalmus occurred suddenly during whooping-cough, a year before coming under observation. The eye was blind, a firm body prevented the return of the globe to the orbit, and a distinct tumor could be felt with the finger on the inner side. After the enucleation a growth about half the size of the eyeball was found on the optic nerve, which was removed and subsequently examined. It proved to be a sarcomatous tumor, probably arising from the fibrous tissue surrounding the nerve-bundles. No recurrence had taken place three months after removal. The author then reviews the literature of the subject : sixty per cent. of the cases occurred under twenty, the tumor was usually included in the sheath of the nerve, and though variable in character was chiefly sarcomatous or myxomatous. In fifteen cases out of sixty-nine attempts were made to remove the tumor without enucleation, in four the operation could not be completed, in five the eye suppurated, and in one death occurred ; the result of trying to save the eye was not encouraging. In the cases cited there were nine recurrences and eighteen were well when last seen. Werner.

Valude (379) reports a case of complete cure, in which four years previously the diagnosis of atrophy of both optic nerves had been made. The patient had been cut with a knife behind the left ear, whereupon sight gradually diminished until he could no longer walk alone. Subsequently a large abscess was found, extending from the mastoid process to Highmore's antrum. After trephining the mastoid he recovered, and V was fully restored. According to the author, there was meningo-encephalitis with descending neuritis. Marckwort.

XX.—INJURIES; FOREIGN BODIES (PARASITES).

380. Briggs, W. E. Notes on the electro-magnet in ophthalmology, with a report of nine cases. *Occidental Med. Times*, Aug., 1889.

381. Lindner, S. Three rare cases of injury to the eyes. *Wiener Med. Wochenschr.*, 1889, Nos. 38 and 39.

382. Marsh, J. P. A case of foreign body in the orbit. *N. Y. Med. Jour.*, Aug. 3, 1889.

383. de Wecker. Les inflammations traumatiques de l'œil. *Ann. d'ocul.*, vol. ci., p. 166, and *Soc. d'opht. de Paris*, March 12 and May 7, 1889.

Briggs (380) prefers a Leclanché battery of sixteen or eighteen cells to develop his magnet. In three of the nine cases only was the operation not successful. Burnett.

Lindner (381) successfully extracted a piece of gun-cap from the vitreous of a young man; he also reported the removal of an eyelash from a traumatic cataract, and mentions a third case in which a small piece of stone had remained in the eye fourteen years. Enucleation was performed, on account of symptoms of irritation, when extensive chalky deposits were found in the phthisical globe.

In Marsh's (382) case a bit of steel remained in the orbit without setting up any considerable cellulitis for sixteen months. It had been probed for often, but unsuccessfully before. It weighed 51 grains, was $\frac{5}{8}$ inch in length, and $\frac{5}{16}$ inch in thickness. The retina was detached from below, involving the macula.

Burnett.

de Wecker (383) opposes combined extraction with very problematical assumptions. According to him, inflammation is synonymous with infection. The injuries of one part of the eye are not more dangerous than those of another, provided infection be avoided. Injuries of the ciliary body are only dangerous when there is incarceration of the iris, causing scars through which infection may enter. These assumptions are energetically opposed in the discussion by Gorecki, Parinaud, and with great force by Meyer. Marckwort.

XXI.—OCULAR AFFECTIONS IN CONSTITUTIONAL DISEASES.

384. Armstrong, S. T. Chronic interstitial hepatitis with hemeralopia, simulating cerebral hemorrhage. *N. Y. Med. Jour.*, May 25, 1889.

385. Despagnet. Rapports entre les maladies des yeux et les maladies du nez. *Rec. d'opht.*, 1889, No. 9, p. 513, and *Soc. Franç. d'opht.*, Aug. 10th.

386. Dillmann, Th. On tabic eye-symptoms. From Schöler's

ophthalmic clinic at Berlin. *Inaug. Diss.*, Leipzig, A. Engelmann, 1889.

387. v. JAKSCH. Multiple disseminate sclerosis in a child of sixteen. *Centralbl. f. Nervenhk.*, 1889, No. 10, p. 318.

388. JOACHIM. On periodic paralysis of the oculomotor nerve. *Deutsches Arch. f. klin. Med.*, 1889, vol. xliv., p. 185.

389. LESZYNSKY, W. M. Localized basal meningitis and double optic neuritis. *N. Y. Med. Rec.*, April 27, 1889.

390. MOELI. On conditions found in affections of the occipital lobe. *Neurol. Centralbl.*, 1889, vol. viii., p. 439.

391. v. MONAKOW. On hemianopsia and alexia. *Correspondenzbl. f. Schweizer Aerzte*, 1889, No. 14, p. 433.

392. MULES. Trophoneurosis ophthalmica. *Brit. Med. Jour.*, 1889, p. 715.

393. NORTHNAGEL. The diagnosis of affections of the thalami optici. *Zeitschr. f. klin. Med.*, 1889, vol. xvi., 5, p. 424.

394. RAEHLMANN, E. On the changes in the retinal blood-vessels visible with the ophthalmoscope in general arterio-sclerosis, with special reference to the sclerosis of the cerebral blood-vessels. *Zeitschr. f. klin. Med.*, 1889, vol. xvi., 5–6, p. 606.

395. SAENGER, A. A case of cortical hemianopsia with autopsy. Essay read at the opening of the new Hamburg Hospital, 1889.

396. UHTHOFF. Investigations on the ocular symptoms occurring in disseminate sclerosis. *Arch. f. Psych. u. Nervenkrankh.*, vol. xxi., Nos. 1 and 2.

397. VERONESE. Report on a case of sudden blindness with normal ophthalmoscopic appearance. *Wiener klin. Wochenschr.*, 1889, No. 24.

398. WESTPHAL. An error of diagnosis in the case of a boy of nine years, who showed the symptoms of disseminate sclerosis. *Charité Annalen*, vol. xiv., p. 367.

399. WIPPLESWORTH. Optic nerve atrophy preceding the mental symptoms of general paralysis of the insane. *Brit. Med. Jour.*, Sept. 21, 1889, p. 648.

ARMSTRONG'S (384) case was that of a sailor, of forty-three years, who had marked hemeralopia, $V = \frac{20}{30}$. No change visible at the fundus. His mind began to wander, convulsions and unconsciousness followed, and finally death ensued. No œdema of the extremities or icterus had been observed during life, yet on *post-mortem* all the characteristics of chronic interstitial hepatitis were found. Meningeal veins congested, but the brain was normal. BURNETT.

DESPAGNET (385) discusses in a very interesting paper the connection between affections of the eye and nose: persistent epiphora, which does not cease even when the lachrymal canal is permeable for the largest probes, frequently depends upon hypertrophy of the lower turbinated bones. The latter had to be treated with the galvano-cautery or saw before the lachrymation came to an end. In conjunctivitis granulosa a new attack of keratitis granulosa may develop, even in the complete cicatricial stage, on account of repeated infection from the nose. According to the bacteriological investigations and experiments of Despagnet, there is a rhinitis granulosa, which bacteriologically resembles trachoma, and is equally contagious. It is very interesting to note that he succeeded in inoculating rabbits with cultures of the granulosa bacillus. MARCKWORT.

Among the one hundred cases of tabes examined by DILLMANN (386), there were forty-one of paralysis of the ocular muscles and forty-two of atrophy of the optic nerve. Syphilis had preceded the affection in sixty-eight per cent. of all cases. Paralysis of the ocular muscles occurred as often in infected as in not infected persons. In studying the figures it must be remembered that the patients were taken from an ophthalmic clinic.

v. JAKSCH'S (387) case was marked by intention-tremor, beginning atrophy of the papillæ, very marked ataxia with enormous increase of the patellar and plantar reflexes. Nineteen cases of disseminate sclerosis in children have been described.

JOACHIM (388) found in 1885 in his patient, a woman, aged twenty-seven, complete right-sided paralysis of the oculo-motor nerve. Attacks of megrim followed, and in December, 1887, again paralysis of the right oculo-motor nerve. He agrees with Senator in assuming periodic or periodically increasing paralyses, but does not consider the former functional disturbances, but ascribes them to pathological changes, especially at the base of the brain.

LESZYNSKY (389) gives the complete history of a case of localized basal meningitis, accompanied with double optic neuritis, in a boy of sixteen, which had been followed closely for five years.

Though there was pronounced swelling of the discs, vision at no time sank below $\frac{20}{70}$, and for the greater part of the time was $\frac{20}{40}$ or $\frac{20}{30}$. The visual fields for white were normal, but contracted for colors. BURNETT.

MOELI (390) finds that in affections of the occipital lobe, beginning during the period of development, degeneration of the optic nerve ensues in man also.

MONAKOW (391) reports on a patient who five years previously had an apoplectic attack, followed by right-sided hemiparesis, right-sided hemianopsia, by alexia and agraphia. The hemianopsia was not complete, as the line of demarcation lay outside the macula lutea. The autopsy revealed various changes, depending upon the apoplexy, and explaining the loss of function. The affection of the gyrus angularis was of much interest, to the degeneration of which, from analogy with other cases, the word-blindness is ascribed.

MULES (392) proposes to include certain diseases under a more comprehensive class of "ophthalmic trophoneuroses." Experiments have shown that passive lesions, such as section of trigeminals, are not of themselves sufficient to produce alteration in the nutrition of the tissues, but that active lesions, such as neuritis, are required for this. The diseases of the eye which the author regards as due to trophoneurosis are: Corneal herpes with neuritis of frontal, lachrymal, supratrochlear or nasal nerves; solitary and multiple phlyctenulæ, corneal ulcers, simple, diffuse, and chronic interstitial keratitis, essential shrinking of the conjunctiva, scleritis, cyclitis, and lastly iritis *e. g.* gonorrhœal and gouty. WERNER.

On the basis of clinical experience and experimental physiological studies of Bechterew, NORTHNAGEL (393) years ago made the following assertion: "When in a central lesion with hemiplegia and paralysis of the facial nerve the arbitrary mobility of the facial muscles is abolished, while the participation of both sides of the face is the same in psychic emotions (*e. g.* laughing), the assumption is justifiable that the thalamus opticus and its connection with the cortex of the brain are intact." In a case which came under his observation the reverse was the case, so he concluded that the thalamus was affected, a conclusion which was confirmed by the autopsy.

RAEHLMANN'S (394) paper is based upon the clinical history of ninety cases, in forty-four of which a pathological condition of the fundus was found. The change in general arterio-sclerosis extends (1) to the arteries. They are tortuous and thin, have a white edge, are contrasted here and there and show aneurisms of the central retinal artery and its branches. (2) To the veins. They have a white edge, are contracted at different places and show varicose dilatations. (3) Phenomena of pulsation are observed, generally in form of locomotions, rarely in form of variations of calibre. The arterial pulse is attributed to the loss of elasticity, extending the pulse into the peripheral ramifications, while the venous pulse (observed in seventeen cases) is brought into connection with the systolic and diastolic difference in pressure within the cavernous sinus. The investigation shows that the participation of the blood vessels of the eye in the similar affection of those of the brain is a common occurrence.

SAENGER'S (395) case was one of typical left-sided homonymous hemiopia, occurring in a man aged forty-six, in which the line of demarcation passed vertically through the point of fixation. The fundus was normal. The patient suddenly died and an autopsy was performed. A circumscribed lesion was found in the cortex of the right cuneus, which extended downward without any distinct line of demarcation into the lobus lingualis. There was also a fresh thrombosis of the basilar artery, and a small lesion was found in the third segment of the right nucleus lentiformis.

After an historical review UHTHOFF (396) gives the clinical histories and records of the autopsies of five patients suffering with disseminate sclerosis, whom he had for a long time under observation during life. He describes very accurately the changes in the atrophic optic nerves, and the difference between them and the tabic, as set forth by Leber and others, and the neuritic atrophies. The condition of the optic nerves, chiasm and tractus is in accordance with the views as to the nature of the disseminate sclerosis of the brain and spinal cord, and agrees particularly with the reports of Charcot and his scholars. It is worthy of notice that in some parts of the optic nerves changes were found which did not differ from these of tabic or neuritic atrophy. There is generally a proliferation of the delicate connective tissue, which can also extend to the larger septa and the inner sheath of the optic nerve. The medullated sheaths rapidly disintegrate, while the isolated or slightly covered axis-cylinders, are frequently preserved. The number of blood-vessels is increased, they are distended and their walls changed. There is generally no secondary degeneration of the fibres of the optic nerve from a lesion. Finally he discusses the different methods of staining. Uhthoff read a paper on the same subject before the Berlin Medical Society (*Berliner klin. Wochenschr.* No. 23), in which the ophthalmoscopic appearances and the visual disturbances are described. Among one hundred cases, changes in the optic nerve were found in forty-five, forty being atrophia. A normal appearance may, however, be found, even when there are very decided changes in the optic nerve and chiasm. In eighteen cases there was pallor of the whole disc, in eighteen of part of it. Pallor is of more frequent occurrence than atrophy in tabes. Total blindness in disseminate sclerosis is very rare. In six cases there was neuritis. The visual disturbances generally assume the form of a relative or absolute scotoma, the periphery of the visual being frequently not at all affected. Concentric limitation was observed in only one case, in which the optic nerve was found normal at the autopsy. In half of the cases the visual disturbance began suddenly, improvement was frequently noticed.

Veronese's (397) patient, a man aged sixty-four, had suddenly become blind without loss of consciousness or disturbance of motility. Ophthalmoscopic condition normal. He had two apoplectic attacks in the course of time, and after one and one half years he died. The autopsy showed chronic leptomeningitis. In the lateral ventricles there was purulent, bloody liquid; optic nerve, chiasm and tractus appeared normal.

Westphal's (398) patient, a boy aged nine, could not look up, and with difficulty to the side; slight ptosis of the eye, nystagmus and atrophic discoloration of the papillæ. Speech slow, trembling of the limbs, marked disturbance of locomotion, increase of the patellar reflex, etc. Diagnosis: disseminate sclerosis. The autopsy showed a tumor in the left thalamus opticus.

Wipplesworth (399) mentions that optic atrophy is usually a late symptom in general paralysis, and relates a case in which it preceded the mental symptoms by some time. Werner.

REVIEW.

HENRY D. NOYES, A.M., M.D. (Prof. Ophthalmology Bellevue Hosp. Coll., Executive Surgeon N. Y. Eye and Ear Infirmary).

A Text-book on Diseases of the Eye, 733 large octavo pages, illustrated by 6 chromo-lithographic plates, 5 plates in black, and 236 wood-engravings. New York, Wm. Wood & Co., 1890.

The book before us is a brilliant testimonial of the extensive knowledge, vast experience, untiring industry, and literary ability of its celebrated author. It presents the actual stand-point of ophthalmology more completely than any other text-book in the English language. Not too voluminous for the student of medicine, it is exhaustive enough for the practitioner who seeks information on rarer forms of disease. Most welcome, however, will it be to the eye specialist. The whole field of ophthalmology, in its various departments, is treated of harmoniously and with singular clearness. Everywhere we recognize the author's desire to do justice to all subjects of scientific and practical importance, but to avoid speculative discussion. The book is truely brought up to the day and the connoisseur readily notices that the author is one of the best-read oculists, his quotations rarely being taken from other text-books, but chiefly from the sources of modern scientific progress, monographs and periodicals.

Dr. Noyes' text-book contains the essentials of the anatomy and physiology of the visual organ, including that portion of the brain which has ocular relations. The methods of examination and the errors of refraction and motility are clearly described. The organic diseases, their medicinal and operative treatment, form the bulk of the book. All the points of importance, especially in the pathology of the inner tunics, the optic nerve and brain, are illustrated by numerous drawings. Due notice is taken of all the advances in ophthalmology, be they in its own sphere or in connecting branches, such as bacteriology, neurology, and general surgery.

We can highly recommend Dr. Noyes' text-book to every one who, in any way, studies or practises ophthalmology.

MISCELLANEOUS NOTES.

THE INTERNATIONAL MEDICAL CONGRESS will meet at Berlin August 4 to 9. The general sessions will occupy three days ; the ophthalmological section will meet on the three intervening days, and the following subjects have thus far been chosen for discussion : (1) trachoma ; (2) sympathetic ophthalmia ; (3) influence of the electric light upon the eye.

DR. DA GAMA PINTO, for many years the assistant of Prof. Otto Becker at Heidelberg, has been appointed Professor of Ophthalmology and Director of the Ophthalmic Clinic of the University of Lisbon.

DR. W. F. DENNETT and DR. J. E. WEEKS have been appointed surgeons at the N. Y. Eye and Ear Infirmary ; DR. CH. F. MAY, Chief of Clinic in the Ophthalmic Department of the Vanderbilt Clinic ; DR. F. HAMILTON WEIR, Assistant Surgeon to the N. Y. Ophthalmic and Aural Institute ; DR. E. FRIDENBERG, Visiting Ophthalmic Surgeon to the Harlem Hospital.

DR. STIRLING D. KENNEDY has resigned the position of Lecturer on Diseases of the Eye and Ear in the Tulane University, New Orleans, and DR. W. C. AYRES has been elected to fill his position.

DR. A. VOSSIUS, of Königsberg, has been appointed Professor of Ophthalmology at the University of Giessen, Germany.

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ARCHIVES OF OPHTHALMOLOGY.

A CASE OF PRIMARY SARCOMA OF THE IRIS, WITH FORMATION OF BONE IN THE SUBSEQUENTLY INVOLVED CHOROID.

(From the Heidelberg Eye Clinic.)

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FORMER HOUSE SURGEON TO THE NEW YORK EYE AND EAR INFIRMARY.

(*With six figures on plates.*)

THE observation long since made, that congenital aggregations of pigment cells have, under certain conditions, undergone development and change of such character as to produce morbid and sometimes malignant processes, has caused surgeons of the present day to look with suspicion on any such accumulation, even supposing the same to occur in a tissue in which pigmented elements exist under normal anatomical conditions. The skin, insignificant in amount as is its pigment in the white races, furnishes not infrequently deposits of the character referred to, v. s., the brown or black mole, the "mother-mark," etc., which, after remaining in a passive state for a period of years, suddenly in middle or later life, due to some unrecognizable influence, take on a change of a malignant nature, demanding surgical interference, with results not uniformly favorable.

The same conditions pertain in the eye, in all the structures of which deposits of pigment cells have been observed. The iris seems, however, the selective ground for their development and growth; here often present themselves small black mounds, emanating apparently from the stroma proper and occupying a plane anterior to the level of the surrounding tissue. These are for the most part benign and remain during life without in any sense interfering with perfect vision; occasionally, however, after years of inactivity, they exhibit changes in appearance and structure, admitting of no doubt regarding their malignancy.

Observations of results occurring sometimes with surprising rapidity, and giving rise in spite of surgical measures to metastatic involvement of adjacent and remote organs, have induced some surgeons to advise the immediate extirpation of such growths wherever found, as the only certain means of avoiding unfavorable possibilities. This rule, while applying, perhaps, for most parts of the body, is subject to an important exception—namely, the eye.

If consulted regarding a perceptible accumulation of pigment upon the iris, which in nowise diminishes the visual acuteness, and which may always remain in an unirritated state, the cosmetic defect being the cause of complaint, can one with entire propriety on such an occasion suggest iridectomy, involving a greater deformity than the existing evil and acting perhaps as a disturbing element in the visual adjustment of the eyes? Could we, however, be absolutely certain that although benign in appearance we were dealing with a malignant growth, there would be no question regarding the imperative necessity for its immediate removal.

It is an unfortunate circumstance that these two varieties of tumors, so essentially different as regards prognosis, present in their primary development decidedly similar appearances, originate in identical elemental tissues, and practically defy differential diagnosis until one has assumed a destructive metamorphosis. The question then arises, is it impossible to distinguish clinically between two growths, the one entirely benign, the other as certainly malignant, before the latter begins to exercise a dangerous influence upon adjacent tissues? This difficult point the surgeon may be called upon any day to decide. The importance to the patient of the accuracy of the diagnosis cannot be exaggerated, for if malignant, even though never so small, its speedy removal is his only guaranty against involvement of other organs.

In consideration of the foregoing facts and of the meagreness of the literature of the subject, the following case is presented, the various details of which appear to offer a relatively complete history of the early appearance, long-continued innocuousness, and subsequent malignant character of such a growth upon the iris; in the hope that the contribution may present some features of interest the writer has ventured upon the appended clinical and anatomical description.

On July 1, 1880, presented himself at the Eye Clinic in Heidelberg Maier N., 49 years of age, from Kaiserslautern, requesting advice concerning a growth upon the iris of the left eye; the history-books of the institution present the following data:

Patient says that the tumor was first noticed when he was ten years of age, being observed by a physician; that in 1867 he consulted Dr. Knapp in Heidelberg regarding the nature of the growth, at that time very small, but which caused occasionally an inflammation of the eye attended with slight hemorrhages into the anterior chamber.

Present Condition.—A dark-brown tumor fills the entire lower inner quadrant of the anterior chamber covering the inferior internal margin of the pupil, which is oblong, no reaction being present owing to posterior synechiæ; the boundaries of the tumor are on all sides sharply defined, only on the outermost edge of the lower portion is seen a root-like projection, a single streak similar in appearance to coagulated blood; the iris has at the outer side a somewhat reddish color; the cornea is slightly hazy from contact with the tumor at several points.

Diagnosis.—Melanoma. Vision, O D $\frac{20}{20}$, emmetropia; O S $\frac{20}{70}$, unimproved by glass.

October 31, 1881.—One year later; tumor appears not to have increased in size. Vision, O D $\frac{20}{20}$, O S $\frac{20}{70}$.

November 13, 1882.—Again observed; tumor decidedly increased in size, spreading forward over the pupillary margin, and extending forward until in contact with the cornea, which is already opaque at this point. Visual field materially narrowed. Vision, O D $\frac{20}{20}$, O S $\frac{20}{100}$. Enucleation advised, but declined.

September 16, 1889.—Seven years elapsed since last examination of eye; patient returns because of unendurable pain for preceding ten days; conjunctiva bulbi markedly congested;

bulbus probably 2 to 3 *mm* more prominent than the right; cornea smooth but quite yellow in color; it is not possible to say whether the substance of the cornea is yellow, or if the discoloration is due to the compact tumor lying in apposition with it posteriorly; toward the outer side and in the extreme upper and inner portion a small area of cornea, 1 to 2 *mm*, remains clear.

Perception of light, lost. Excursion of bulbus undiminished. Tension increased. Vision, O D $\frac{20}{20}$.

September 19, 1889.—O S enucleated.

September 26, 1889.—Wound well healed and patient permitted to leave institution with instructions to use boracic-acid solution for cleansing.

October 20, 1889.—Prothesis.

Patient stated in his first examination that he had consulted Knapp in 1867, and in "Intraocularen Geschwülste," page 220 (English translation, p. 299), is found the following description of what may be fairly inferred to be the foregoing case:

"The growth concerned a man from Kaiserslautern, thirty-one years of age, who presented himself for treatment. Fifteen years previously he had noticed a small tumor in his left eye which in no respect disturbed vision; he experienced no inconvenience from the same, save that occasionally every three or four months during the last few years there had appeared slight hemorrhages into the anterior chamber, which rapidly reabsorbed. I found upon examination an eye normal in structure, appearance, and function. In the lower inner section of the iris and in the large circle were situated beside one another three partly round, grayish-brown, velvety-looking tumors, of which the two larger possessed an apparent diameter of about 5 *mm*, the smaller growth about 3 *mm*.

"The portion of the iris lying between the growths and the pupillary margin was grayish-brown pigmented and pale, while the remaining iris was of a glossy, light-brown color. Between the growths and the insertion of the iris lay a strip of tissue which was likewise pale and brown pigmented.

"Beside these growths there were to be seen in the lower inner iris segment in the middle of the large circle three pale-brown spots, slightly separated from one another and in size scarcely as large as pin-heads; these have quite the same appearance as the previously mentioned larger growths and must be considered of similar structure—it was not possible definitely to determine the presence of vessels in any of these tumors."

We have here presented a case whose history is complete, dating from a time when, in the tenth year of the patient, the pigmented tumors were noticed with fair probability that they were congenital, as small dark lumps, in nowise obstructing vision, seen later as middle-aged man when the growths had formed a very slight visual impediment with accompanying hemorrhages, observed at subsequent periods when the sight was materially diminished, and finally demanding treatment when the function of the organ had been entirely lost.

A comparison of the anatomical structure of those growths, proved upon microscopic examination to be true melanomata with that of tumors whose early appearance suggested benign properties, but which in the course of years have taken on a malignant character, may present contrasting phases of interest.

Unfortunately for our anatomical knowledge of iris melanoma, the literature of the subject is very meagre, since the number of such growths, anatomically examined, is exceedingly small. Knapp, in "Intraocularen Geschwülste," offers the following brief description: "The tumor consists of a circumscribed enormous development of partially unpigmented but for the most part pigmented stroma cells of the iris; these cells are ramous and anastomotic; between them and the adjoining tissue there is no definite boundary, and the remaining portions of the iris are normal."

Another case of true iris melanoma was examined by Fuchs (*Arch. für Augenheil.*, vol ii., p. 435). He observed a dark-brown roll at upper inner quadrant lying in pupillary space and apparently emanating from the posterior surface of the iris. On use of atropine pupil dilated readily and symmetrically, showing that the growth occupied the greater portion of the superior internal quadrant of the iris, and was mainly in the posterior chamber; the remaining iris was quite normal. Patient, seventy-five years of age, was unaware of the presence of the growth, having consulted the surgeon with regard to failing vision due to immature cataracta senilis; growth, removed by iridectomy, presented the following structure macroscopically: A dense, nearly evenly, colored black mass with moderately sharply defined borders; no details could be determined. Microscopically the cells constituting the growth are seen heaped together, and containing such quantities of pigment as to render it impossible to recognize the presence of nuclei; where the growth borders upon physiological iris tissue these cells are found containing less pigment, and their structure can be determined. They seem similar in all respects to those normally constituting the stroma of the iris, with the addition of an unusual amount of pigment; no trace of blood-vessels or of any connective-tissue stroma could be found; in other words, the anatomical structure is that of an absolutely benign growth, in which are contained no elements not normally existing in the tissue in which it is located.

Knies (*Arch. f. Augenheil.*, vol. vi., p. 175) mentions a case in which in an eye enucleated for melano-sarcoma there was found a small melanoma of the ciliary processes lying between lamina elastica choroidea and pars ciliaris retinæ; the surrounding choroid was perfectly normal, uninfiltrated by any cells from the sarcoma lying at the posterior pole of the eye. Macroscopically these growths appeared essentially the same, differing of course in size. Microscopically the one consists of nothing but large cells of the choroidal stroma, densely pigmented, heaped upon one another, *i. e.*, a true melanoma; the other exhibits the characteristic structure of a sarcoma, vascularization, inflammatory fibrous stroma, and the usual cellular elements of such a neoplasm.

That two growths in their elemental structure identical should in the course of development assume such diverse qualities is difficult to realize; evidence is, however, not lacking to corroborate this view. Cases of melanoma, which after years of quiet occupancy of tissue have involved the organ in a destructive inflammation, are reported by Hirschberg (*Graef. Arch.*, vol. xiv., p. 285), Warren Tay (*Ophthal. Hospital Reports*, vol. v., p. 230), Schies (*Virchow's Arch.*, vol. lxix.), and Hosch (*Centralblatt. f. pract. Augenheil.*, 1881, p. 361).

The anatomical examination of a fifth such case is here appended somewhat in detail.

Macroscopic examination (see Fig. 1, and its explanation at the end of this paper).

The enucleated globe presents externally nothing of pathological interest, being thoroughly symmetrical in form, with a normally glistening white sclera, to which in points the capsule of Tenon adheres; cornea is a dirty yellowish color; optic nerve of ample length.

Having been hardened in the usual chromic-acid and Müller solutions, the eye was divided through its horizontal meridian as nearly as possible in the centre of nerve and cornea: the color of the contents of the globe is dark-reddish brown; the various chambers of the organ, anterior, posterior, and vitreous, can no longer be recognized as dividing boundaries, being of uniform tissue structure; the sclera seems, especially toward the posterior pole of the eye, to be materially thickened, but closer examination discovers this apparent hypertrophy to be an involvement of the choroid, which is through inflammatory cellular and fibrous invasion many times its normal thickness and density; it has as well over its entire surface grown firmly adherent to the sclera. At that portion of the eye in which the ciliary body and iris are normally located, there is presented an ill-defined crescent, nearly parallel with the corneal contour and in apposition with it, consisting of very black pigment; of the lens there is seen absolutely no trace.

The tumor mass, owing to several large hemorrhages, has a somewhat peculiar shape, the form being not dissimilar to that of a very low-spreading tree, whose trunk, starting from optic-nerve entrance, speedily puts out drooping branches to either side, extending as far as the sclera; the top of the tree as it approaches the cornea, instead of being pointed, spreads out to either side of a large hemorrhage which occupies nearly the position of the lens when in place; at the back of the eye, at either side of the trunk of the tree, are two other large extravasations of blood; these furnish the impression of being recent, as they are of a deep reddish color. There are many other hemorrhages in the tumor, insignificant, however, in comparison with the above-mentioned.

Both halves of the globe were examined with the following microscopic finding:

The corneal epithelial layers were, with the exception of the innermost columnar, quite normal. This latter presents certain interesting anatomical changes, which may have been factors in causing the yellowish discoloration observed before removal. This inner layer is in several places so swollen as to quite destroy any distinctness of outline between the cells, and there are points where pressure has caused their entire degeneration, the nuclei being no longer seen and a granular detritus occupying the location of the former healthy cells; this condition is observed with greater or less frequency over the full extent of the cornea.

The epithelial covering of the cornea is, in consequence of the presence of a nearly white fibrinous structure, separated throughout its area from the underlying Bowman's sheath for a space corresponding in width to several times the usual thickness of this membrane. The substance filling the above-mentioned space has entered from the deeper tissues of the cornea, through numerous points of rupture in Bowman's membrane, and through inflammatory exudation from the engorged vessels at the limbus corneæ. These latter, especially in preparations somewhat remote from the horizontal meridian of the eye, are greatly distended with red and white blood cells, the latter of which, in profusion, migrate in all directions from the thinned vessel walls.

The structureless substance, filling the artificial chamber mentioned, remains unstained by ordinary reagents and is most likely fibrin. There are found embedded in it numerous cells, mainly of a small round variety, which can be seen entering this enclosure at the points of solution in Bowman's membrane, to which they have made their way from the cornea, their presence in which will be explained below, and spreading themselves about in irregular heaps, with occasional long lines lying parallel with the limiting border of the containing space. These cells are in all respects similar in appearance to those composing the bulk of the tumor, and together with leucocytes and a few of the larger tumor elements comprise the contents of the space. That no connective-tissue cells are here found, the fibrin being not yet organized, may be considered an indication that the spread of inflammatory action had but recently extended so far. It has, however, been sufficiently severe to cause degeneration and also hypertrophy of the corneal epithelial layers. Bowman's membrane where destroyed has become frayed, giving the appearance of embracing the cells in a delicate stroma (see Fig. 2), but few vessels are contained in this exudation, and such as are present are in the immediate vicinity of the limbus.

The cells of the neoplasm situated in the cornea are found most numerously in the centre and to the inner side. They can be traced directly from the remains of the iris through Descemet's membrane into the corneal substance,—a dense inflammatory mass taking this direction, enclosing in its meshes multitudes of neoplastic cells in various stages of development.

Descemet's membrane is greatly hypertrophied, to at least double its usual thickness, and its continuity is ruptured in many points, owing to ulcerative processes. The result of these rents has been that the elasticity of the structure has caused it to draw away at either side of the breaks and double upon itself in wave-like folds or loops, which are in turn often split in the long axis of the tissue (see Fig. 3). These splittings or clefts serve as nests for the accumulation of cells, which are present in all in greater or less abundance, now and again destroying the apex of the loop and establishing a connection with the tumor mass in the globe of the eye.

Through the largest of these breaks in Descemet's membrane enters, nearly midway between corneal centre and limbus, a compact fibrous structure, already mentioned, which, directly it comes in contact with the cornea, spreads out at either side and insinuates itself into the tissue until differentiation is no longer possible (see Fig. 3).

These fibrous bundles contain cells in great numbers, and to this origin is mainly due the presence of those found in the cornea. They are likewise rich in vessels, containing many from the iris, with their normally thick walls much hypertrophied; also numerous fine vessels of new formation with delicate walls and large endothelial cells.

That this perforating growth is the remains of a portion of the iris is rendered more probable by the presence of a considerable number of pigment cells very like those of the normal iris stroma, in addition to which there is much degenerated pigment. These cells have insinuated themselves into the corneal tissue, and in spaces between the trabeculæ of this structure are found in small aggregations, heaped together without any evident attempt at arrangement. On sections made nearly through the horizontal diameter of the cornea the canal of Schlemm is to be found at the outer side with walls much compressed and infiltrated, still, however, to a certain extent patulous, while at the inner side there remains no trace of the sinus, its normal site being a compact accumulation of cells.

In sections more remote from the horizontal meridian of the eye, the severity of the inflammation appears much aggravated, in consequence of which the tissue at sclero-corneal margin has undergone such change as no longer to be recognized as characteristic. The structure is here a very delicate but well defined stroma, nearly areolar in appearance, in whose meshes are cells, the majority small and round, associated with leucocytes and various other larger cellular elements containing nucleoli. At this portion of the sclera are seen vessels whose distended walls, engorged with blood, contain also in their lumina small bodies which are microscopically in all respects similar to those of the neoplasm, and present in too great numbers to have been carried into the vessels by mechanical violence. These vessels are from both sclera and conjunctiva, and there is hardly a reasonable doubt that at the sclero-corneal junction the growth has penetrated the coats of the eye and involved the conjunctiva. So far as can be determined, this infiltration extends but about one millimetre from the limbus, and the margin of the conjunctiva, divided in the operation for enucleation, appears cell-free.

The lymph-spaces of the cornea and sclera are much enlarged, and serve in many cases as receptacles for cells, which seem to collect by preference in such locations. At the limbus these spaces are so materially widened as to appear like sinuses or large vessels cut in a very oblique direction; the absence of any regular wall, however, dispells at once this suggestion, and the idea appears tenable that the wide spaces may be due to an absorption of surrounding scleral tissue by the densely packed cells here accumulated. An index of the intensity of the inflammation at these points may be found in the proportionally great profusion of small round cells.

At some distance anterior to the equator of the globe the infiltration of the sclera rapidly diminishes, and posterior to the equator is no longer present; the tissue presenting a normal appearance, and offering no inflammatory evidence, save on the inner surface, where it has grown fast to the choroid.

The tissue of the iris has, during the slow development of this growth and the progress of the inflammation, suffered to such an extent that its remains are no longer to be recognized as possessing any histological similarity with the original structure. We find directly in apposition with and closely adherent to the thickened Descemet's membrane a body occupying the anatomical location of the iris, anterior and posterior chambers of the eye, consisting mainly of a dense stroma, rich in vessels with thickened walls, and supplied with a plentiful amount of pigment-cells; in addition to which there are many small, nearly round lumps of inky black granular pigment, the possible relic of the uveal lining of the iris. In this tissue there are neither depressions nor inequalities, muscular cells from the sphincter, or aught else to indicate the site of a previously existing pupil. Nevertheless the location renders it moderately certain that this altered structure was formerly iris. That absolutely no trace of the normal anatomical structure of the latter should remain is scarcely to be wondered at, although no suppurative action was ever established, when it is remembered that this tissue was for twenty odd years the seat of inflammatory action sufficiently severe to give rise to frequent hemorrhages. That the entire structure was not at once involved is true, but the process increased latterly in intensity sufficiently to disintegrate in a short time a tissue displaying such delicacy as the iris.

The changes in the ciliary body are scarcely less extensive than those above mentioned. The processes at the inner side have been materially displaced backward toward the equator of the globe, by the pressure of the growth advancing in this direction; while at the outer side the corresponding portion of the ciliary muscle is drawn forward toward the corneal centre. This traction is caused by the infiltrated zonula acting as a pulley; pushed backward and downward at the inner side, the outer has been drawn inward with it (see Fig. 1).

The contents of the ciliary region have undergone extensive alteration in such a degree as to render their recognition microscopically difficult. There is, however, a kind of order in the areas of connective tissue, which suggests the normal arrangement of the ciliary muscular fibres, while now and again the long nuclei of the unstriped muscle can be seen. These are, however, so easily confounded with the long slender nuclei of new-formed inflammatory tissue which here abounds, that they are not to be determined with any great degree of positiveness. The enlarged ciliary processes are composed of connective-tissue striæ following the direction of the meridional muscular bands. The meshes of this radiating web are filled with the several varieties of cells composing the growth. As far back as the ora serrata the ciliary body seems evenly infiltrated, but from this point to the posterior pole of the eye the intensity of the process is much less marked, and although changed decidedly these tissues, retina excepted, still retain many normal histological properties. The vessels of this region are dilated and very full of blood, showing occasionally where, owing to excessive engorgement, the weakened and distended walls have ruptured and the contents been effused into the surrounding tissue, making small sinuses, which are apparently limited by a border of neoplastic cells, and in some instances by a wall of fibrin. The uveal layer of the ciliary process and the pigment-cells of these organs have undergone hypertrophic change, so that the amount of pigment not elsewhere excessive is here very great, and disposed about in disordered collections like a thick sooty deposit; much of it is disintegrated, masking the structures which underlie it. The remaining portions of the choroid exhibit similar appearances, the layers being occupied by what seems to be an extraordinary number of vessels; this vascular richness is probably due not to greater numbers, but to the fact that the engorgement renders visible and even prominent capillaries usually not observed in the normal choroid.

The many varieties of cells to be found in sarcomatous growths, and the proneness of the latter for producing tissues histologically remote in structure from those in which they are situated, is well illustrated in the eye under examination by an example of such rare occurrence as to merit special attention and description. The anomaly referred to is a small mass of true bone deposited in the choroid at the posterior pole of the globe, in such a position as to form at the choroidal ring a closely fitting, somewhat constricting collar about the optic nerve, by which it is pierced eccentrically.

As nearly as can be determined, the bony substance lies between the chorio-capillaris and the sheath for the greater vessels (see Fig. 4). This conclusion is favored by the fact that it is bounded internally and externally by choroidal layers containing large vessels and pigment in abundance. That the bone deposit originates in the chorio-capillaris is rendered probable by the presence on its external border of what appears to be a definite limiting membrane of fibrous tissue quite unbroken and not participating in the process of ossification. Between this membrane and the sclera, the supra-choroidea and sheath for the greater vessels lie comparatively free from connective-tissue changes; the internal and lateral margins of the bone are interrupted at short intervals by deep indentations containing penetrating bundles of incompletely ossified fibrous tissue. The transformation of this newly formed inflammatory tissue into bone is plainly seen at many points along the inner border of the mass: the connective tissue as it approaches the bone becomes gradually more dense and nearly homogeneous in appearance, still nearer it receives a deposit of lime, which rapidly ossifies, surrounding the former connective-tissue nuclei and transforming them into perfect bone-cells; here and there can be seen nuclei partly enclosed in bone and part lying free in the adjoining inflammatory tissue, the bone growing not alone at the periphery but upon the internal surface as well. The nourishment of this structure is amply provided for by numerous large vessels traced directly from the choroid, some presenting circumferences not entirely enclosed in the encroaching growth, which abounds as well in marrow-spaces containing large vessels in the Haversian canals.

At infrequent intervals there are found, in the bone, nests of the larger sarcoma cells, in no case lying far from the margin, and having a small opening leading to the adjacent tissue, from which they have wandered into their present abode.

Although the osteoid deposit projects at either side sufficiently to constrict the nerve, in none of the preparations of the eye is it found that any portion of the former is included in the process of ossification. The bone is in contour a nearly round shell, convex on its surface in apposition with the outer choroidal layers and slightly concave on its inner aspect.

A highly magnified section shows this to be a deposit of true bone containing all the histological elements of such tissue when found under normal relations (see Fig. 5). There are narrow spaces containing Haversian canals holding in their centres large perfectly formed vessels with well developed endothelia. The vessels are filled with blood, furnishing conclusive evidence that the canals performed the same nutrient function here as in normally existing bones elsewhere, and that they are not mere sinuses; fat-cells are present in the marrow spaces in abundance, while about the borders of said spaces osteoblasts are seen in process of calcification. Here are also found the giant-cells or osteoclasts, possessing several nuclei, and indicating the presence of a process of rarefication plainly observed in the uneven outline of the periphery of the containing spaces, as well as at *H.* (see Fig. 5), where the intervening substance has been absorbed, causing a connection to be established between them. Whether or not a formation of new bone follows this process, was not to be determined in any of the preparations.

In the compact substance of the bone is clearly seen the concentric distribution of the Haversian lamellæ, the lacunæ with anastomotic canaliculi containing bone-cells which were formerly the nuclei of the uncalcified connective-tissue fibres; in fact, a section of normal tissue found under conditions where all other structures are abnormal,—"a result," says Arnold, "only to be obtained upon a basis of connective tissue, upon which under the influence of sarcoma any tissue foreign to the location may develop."

That this mass is the result of an ossifying process in fibrous tissue is certain, inasmuch as the most thorough scrutiny fails to establish the presence of any cells which are beyond doubt cartilage, although there are, as previously mentioned, osteoblastic cells in the marrow-spaces undergoing calcification, which are in no respect similar to connective-tissue elements. These are, according to Stöhr, embryonic cells with a tendency to bone formation, which deposit themselves by preference at points involved in such metamorphosis. Regarding the conversion of connective tissue into bone the author mentioned says: "Single connective-tissue fibres become calcified, and on these are deposited osteoblasts, originating from embryonic cells, forming, after the manner described, bone."—Stöhr, 1888, p. 69. Pagenstecher, *Graef. Archiv*, vol. vii., p. 112, regarding this subject, says: "Simple calcification of connective tissue presents only a passive process of retrograde metamorphosis. Transition into bone-formation is to be considered a decidedly active step. Throughout there must first be present, under co-operation of cellular structures, a special metamorphosis, and wherever this is incomplete the process advances no further than simple calcification. The development of bone from cartilage does not occur under such conditions." The process is essentially similar to the bone-formation from periosteum.

The occurrence of deposits of bone in the eye is by no means so rare as to occasion special interest. So many phthisical globes, having lost their function through plastic irido-choroiditis, have developed bone substance that its presence under such conditions excites no remark. That the choroid is in most if not all cases the source of these formations, the observations of Pagenstecher and Knapp have definitely determined, the latter maintaining that ossification can only take place in other tissues of the eye into which exudation from the choroid has spread. He finds bone to originate in all cases in the chorio-capillaris by virtue, as he thinks, of the great vascularity and nutritive powers of this layer. While agreeing with Knapp as to the choroidal origin of such growths, Pagenstecher considers the source to lie, in the majority of cases, in the glass lamella.

The appearance of bone in the eye in the progress of a sarcomatous inflammation is a phenomenon of such infrequency that until 1883 no case of the kind had been reported. In that year Leber presented at the Ophthalmological Congress in Heidelberg (see *Zehender*, 1883, p. 193) microscopic sections of a sarcoma of the choroid, in which was to be seen a shell of true bone. This, he said, was, so far as his knowledge extended, "an ophthalmological unit." He recites the history of the patient in connection with the case, and says that, although no injury was acknowledged, there was a perforating scar at the sclero-corneal junction, adhering to which was a portion of the ruptured lens capsule. The eye had never been subjected to an operation. A search for a foreign body resulted negatively. He gave as an opinion that the eye had received a punctured wound. The microscopic examination of this same case is reported in detail by Nordensen (*Graefe's Archiv*, vol. xxxi., 2, p. 59). His description would indicate that the growth was a true osteo-sarcoma, or at least the nearest approach to that tumor of which we have any connected report. Other cases are found in the literature, but with such meagre attention to anatomical examination as to render them practically worthless as standards of comparison.

Alt gives, in his "Compendium der Histologie des

Auges," an illustration of bone structure which he says he had observed in a sarcomatous growth of the eye. No description, either clinical or anatomical, is offered, the statement merely being made that such finding is rare.

Fuchs, in "Das Sarcom des Uvealtractus," 1882, says: "It is exceedingly doubtful if a true osteo-sarcoma of the choroid has ever been observed."

In our case there can be no doubt of the presence of true bone, situated in tissues undergoing a sarcomatous process, and originating in connective tissue, in whose meshes were contained cells of the malignant growth, which in many cases are found surrounded by the bony structure, giving the appearance of cell nests. The fact of these cells being found within the continuity of the bone would perhaps indicate that the ossified tissue was entitled to consideration as a process established in the changed connective tissue of the choroidal stroma, and not the simple calcification of an inflammatory exudation.

The retina, owing to its delicate texture, has undergone such extensive changes during the progress of the inflammation as quite to have lost all traces of normal anatomical structure, the only recognizable remaining portion being the nerve-fibre layer, and the distinguishing feature of this is its direct connection with the optic papilla. Histologically it retains none of its former properties; the remaining retinal layers have become so thoroughly filled with fibrous tissue and other products of inflammation that their situation is to be but relatively determined. Probably the delicate, slightly pigmented membrane overlying the choroid (see Fig. 4) at either side of the nerve entrance, and supporting the richly infiltrated nerve-fibre layer, contains the remnant of this structure.

The cellular infiltration of the retina is, with the exception of the nerve-fibre layer at the posterior pole of the eye, immaterial. In this last-mentioned layer, however, are found sarcoma cells, with wandering leucocytes and pigment elements in abundance. There are present here as well the very large irregular-shaped cells, found for the most part in the vitreous, and at such places only as are already in a degenerative stage. In the stroma of iris, choroid, etc., these cells are wanting; only the small, round, rapidly proliferating elements associated with fibrous-tissue cells, and leucocytes are found. That these large cells are confined to the area of the vitreous chamber may perhaps be explained by the fact that floating free in the fluid vitreous they have grown rapidly in the absence of pressure, and have as quickly, owing to insufficient nutriment, degenerated, while the other cells invading the vascular tissues have occupied sufficiently nutritive areas to retain their aggressive qualities and reproduce themselves extensively.

The intraocular portion of the optic nerve presents, under a low magnifying power, a very decided enlargement, projecting considerably into the eye. To mechanical causes are attributable these changes in structure and outline. The process of connective tissue formation has here been very active, and numberless bundles of the same have arranged themselves in a peculiar and almost grotesque manner. They lie superimposed upon and interlacing with one another something after the fashion of a coarsely woven texture (see Fig. 4). In this tangle are numerous colonies of cells, the majority of which belong to the fibres of the disproportionately abundant inflammatory tissue. The infiltration appears to extend backward in the nerve only so far as the lamina cribrosa.

The forward projection of the optic nerve is caused by the presence at either side of the choroidal ring of the bony deposits with overlying connective tissue, rendering it impossible for the nerve to assume its normal position. The arteria centralis retinæ displays a considerable hypertrophy of its walls, but no material cellular infiltration of adjacent tissues.

The fibres of the optic nerve, stained by Weigert's method, are found completely atrophic. The sub-vaginal space shows moderate infiltration.

Several interesting features, both anatomical and topographical, are presented by the lens as the result of its participation in the destructive process here enacted. The luxation first attracts attention because of its direction and extent, which are so considerable that upon division of the globe in the horizontal meridian there is macroscopically no trace of the lens to be seen. The upper half of the eye, first prepared and examined microscopically, contains no tissue presenting a structural similarity to that of the lens. Altered ciliary processes are found in all sections, but nothing to be recognized as zonula.

In preparations from the lower half of the eye the remnant of the lens is discovered dislocated backward, downward, and inward in such a direction that a line drawn through its long axis meets at an angle of forty degrees a line drawn parallel to the antero-posterior diameter of the globe; the latter line passes through the zonula at a distance of several *mm* from the upper and outer extremity of the lens, the superior border of which occupies a level below the centre of the rectus internus muscle.

The angle of displacement is doubtless due to the direction taken by the growth after involving the ciliary body. Seven years previous to enucleation it had advanced into the anterior chamber, inferior internal quadrant, sufficiently to cause a decided haziness of the cornea with which it was already in contact. After reaching this resisting structure as a forward limit, the growth would naturally proceed in the line of least resistance, which must be over the tissue of the uninvolved iris and backward along the ciliary processes. This backward progress has pushed the zonula, at the inner side of the eye, before it, together with the ciliary processes in the same meridian, until they occupy a relatively greatly changed level in comparison with their normal situation (see Fig. 1). The zonula to the outer side, affected at a later date by the growth, is drawn by the dislocated lens somewhat forward and slightly toward the centre of the cornea. The displacement is not so great, nor is its tissue degeneration equally decided with that of its corresponding structure at the inner side, which contains throughout little else than compact bundles of fibrous tissue.

The suspensory ligament at the inner side must long since have been ulcerated through or ruptured by pressure; in any case the remains of the shrunken capsule are in direct contact with the ciliary processes at a point nearly opposite the centre of the lens (see Fig. 1). The anatomical changes sustained by this structure during the progress of the tumor are considerable, concerned, however, principally with the capsule and cortex, the nucleus being, with the exception of minute alteration, nearly or quite that found upon removal of an ordinary senile cataract. The capsule of the lens, ruptured in many points, large sections being often entirely wanting from absorption, has drawn away from the points of solution in its continuity owing to the elasticity of its structure, and curled upon itself in loops. At the outer side of the lens the capsule, while irregular in outline, has not at any point been broken through, and can be found in all sections intact, exhibiting the same decided increase in thickness with other parts of this membrane. The cellular elements of the cortex are nowhere to be recognized, retaining no structural similarity with the original substance. At the outer side, where the capsule remains unruptured, is observed a loss of cortical substance, having a round opening lined with such material as is usually found in cystic degenerations. In the adjoining cortex are seen cells strikingly similar in appearance to the bone cells encountered in the already described deposits in the choroid. That they are bone is improbable, and not susceptible of proof. They suggest the possibility of the presence of a calcifying process, which has not advanced to the stage of ossification.

Through points of rupture in the capsule the cells of the surrounding growth have made their way in great numbers. The presence of many engorged vessels and sinuses in the immediate vicinity furnishes a vast supply of leucocytes, which wander in colonies in all parts of the lens, nucleus excepted, this remaining free from cellular infiltration.

The remains of the lens, around which the capsule has contracted as the cortex gradually underwent degeneration and absorption, are in amount scarcely one half that of a normal structure. The vitreous chamber contains the bulk of the tumor, the outline of which, as previously described, is in certain respects similar to that of a low-spreading tree.

In cellular elements the growth consists of three varieties or sizes, representing different grades of development; these are suspended in what appears a homogeneous supporting matrix, lacking in stroma, or if such be present it is of so delicate a texture as to be scarcely recognizable save where it borders upon tissue, like choroid, in which a stroma has formed from hypertrophy of previously existing connective-tissue.

The vast majority of the cells are very small and round, with but an insignificant quantity of protoplasm containing a nucleus. A slight advance in development shows them with a greater amount of protoplasm, in which the nucleus is often eccentrically situated; these are found in greatest abundance where the progress of the growth is most pronounced and rapid. The adult sarcomatous cell is mainly oval rather than round, and contains several nucleoli. The third variety is the beginning of a degenerative stage: the cells, greatly increased in size, gradually lose their distinctness of outline, nucleus becomes granular and partly masked in the disintegrating protoplasm, and finally the cells melt down into an amorphous substance in which other cells float about; small highly refracting points indicate the presence of a fatty metamorphosis.

There are many dilated vessels and sinuses in the vitreous portion of the tumor, in which are found cells undoubtedly of the same variety as those comprising the growth; they have been carried to their location by the rupturing into one another of small vessels from absorption of intervening walls. It is hardly probable that circulation has been maintained in any of these channels after the entrance of the cells, for in such case the entire economy of the patient would be liable to metastatic affections.

Two large hemorrhages, surrounded by a thick border of cells, find place in the growth. These furnish the impression of being of recent formation, since the blood corpuscles are distinct in outline, in nowise degenerated, and enclosed in no limiting fibrous membrane, such as is found in many of the neighboring small extravasations.

A fact which lends weight to the theory of recency in the above process is that the patient was seized, a few days previous to enucleation, with sudden and unendurable pain, attended with decided increase in the tension of the globe. He was thus compelled by pain to seek surgical aid, of the necessity for which entire loss of vision had failed to convince him. That primary sarcoma of the iris belongs to the more infrequent of tumors is demonstrable by a casual inspection of the literature of diseases of the uveal tract. Knapp's work, "Intraocularen Geschwülste," 1868, may be considered as the inaugural effort in an era of classification of tumors. In this volume, p. 175, he says: "Primary sarcoma of the iris is not, in my recollection, an existing disease." Since that date, however, a considerable number of such tumors have been reported. Fuchs, in "Das Sarcom des Uvealtractus," 1882, has tabulated from various sources sixteen cases, fourteen of which were anatomically examined; eleven additional cases are now contained in the literature, making twenty-seven in all.

Five of these have supervened upon simple melanomata of the iris, which were noticed in early childhood, with strong probability that they were congenital, remaining for many years unirritated, and later without attributable cause involving the organs in a destructive process.

The clinical history of this variety of cases suggests an argument in favor of the theory of Cohnheim regarding malignant growths; for here appears a certain excess of embryonic cells above what are structurally of use, heaped together in a tissue whose anatomical elements are identical with such accumulation.

Of the utmost importance is it to discover, if possible, some means of early differentiation between growths which remain always benign and those which in course of time become malignant. In the earlier stages of the pigmented growths of the iris this is by no means easy. Having a common origin in the stroma cells of this structure, these two tumors present appearances of such decided similarity that an absolutely certain diagnosis is often only possible after an extended period of observation.

The remarkably small number of melanomata removed bears eloquent testimony to the innocuousness of this growth; its tendency is to remain passive. Hence it happens that the attention of the patient is sometimes first drawn to the presence of the growth by associates detecting the cosmetic defect; subjective symptoms were entirely absent. For a certain period in its history sarcoma follows a like inoffensive course, and perhaps the first deviation to be noticed is its sudden tendency to growth and vascularization, simultaneously with which, pain, hemorrhages, failing vision, or other subjective symptoms appear, whose presence may be practically accepted as sufficient warning that the hitherto quiet resident has become a dangerous tenant, and that the moment for considering the advisability of removal has arrived. Fortunately it happens that up to this stage in the development of tumor the results of operative procedure in sarcoma of the iris are exceedingly gratifying, in all cases where an early iridectomy was performed, both as regards vision and immunity from recurrence. Eleven cases thus treated furnish the following data:

2 cases previous to operation,	$V = \frac{20}{20}$;	subsequent,	$V = \frac{20}{20}$;	no recurrence.
1 case “ “ “	$V = \frac{20}{100}$;	“	$V = \frac{20}{100}$;	“ “
1 “ “ “ “	$V = \frac{12}{30}$;	“	$V = \frac{12}{40}$;	“ “
1 “ “ “ “	$V = \frac{20}{70}$;	“	$V = \frac{20}{200}$;	“ “
1 “ “ “ “	“blindness”;	“	$V = \frac{20}{30}$:	“ “
3 cases vision not mentioned	———		———	“ “
2 “ “ “ “	———		———	recurrence.

Thus of eleven cases operated upon, nine resulted favorably; three are mentioned with no recurrence, vision being omitted. The reports of these were available only from annual reviews, which were too brief to be of much value. In both cases in which the growth recurred iridectomy was of decidedly questionable expediency, and the result should not be permitted to militate against a favorable consideration of the operation; in the one, both irides were involved in the malignant process previous to operation; in the other, the growth had already extended behind the sclero-corneal margin, and there was recurrence in the corneal wound. Enucleation showed that the growth involved the zonula

Zinnii. The operation could not therefore hope for a successful issue.

A glance at the visual result shows that eyes possessing normal vision previous to operation, retained the same. Those with slightly diminished vision experienced a further almost immaterial diminution. The case marked "blindness" was complicated with glaucoma, and an unusually favorable result, $\frac{20}{30}$, followed the iridectomy consequent upon removal of the neoplasm.

The healing subsequent to operation was unattended by inflammatory action, save in one instance, when an iridocyclitis diminished vision from $\frac{20}{70}$ to $\frac{20}{200}$, the eyes, however, remaining healthy as long as observed, about one year. The period of observation after operation continued from six weeks to three years.

Iridectomy appears to have every thing in favor of its early employment. The iris being contiguous in tissue structure at but one point is particularly favorable for removal of any growth contained in its meshes, and enucleation can always be resorted to in the event of recurrence.

When we note that of the twenty-eight melanotic tumors of the iris examined twenty-six are malignant, there appears reasonable ground for regarding with suspicion all such growths. They should be considered as intruders, to be tolerated only as long as they display an absolutely passive disposition, any indication of irritability being justification for removal. Should temporizing measures be employed until a diagnosis of malignancy can be clearly established, the time for a minor operation may be already past and grave consequences at hand (case of Hirschberg, *Graefe's Arch.*, vol. xiv., 3, p. 285; enucleation, for sarcoma of the iris, of an eye having $\frac{20}{30}$ + vision).

The tumor whose anatomical examination we have here offered must, owing to the stage during which it was removed, be regarded for purposes of comparison as a choroidal growth only, notwithstanding it had presented during its development three distinct phases: observed for twenty-five years as a harmless accumulation of pigment cells offering not the slightest inconvenience; for

Fig. 1.

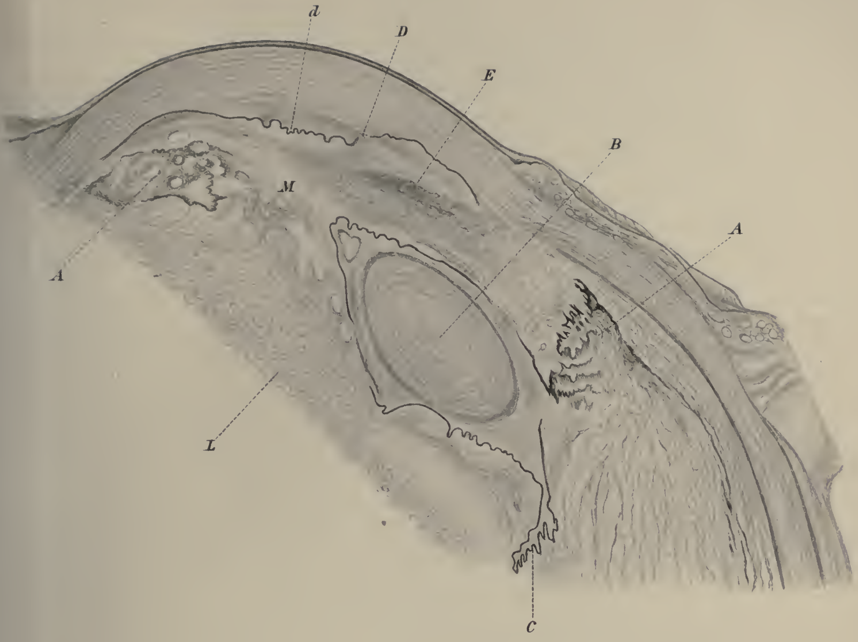
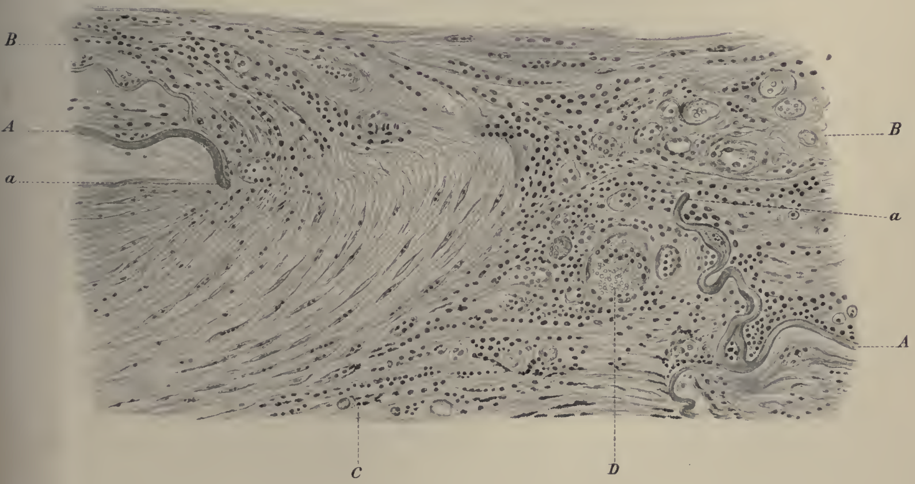


Fig. 3.



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thirteen years as a slowly enlarging tumor of the iris, causing comparatively frequent hemorrhages, plastic iritis with posterior synechiæ, etc.; and for seven years as a growth regarding whose malignancy no doubt was entertained. Particular interest attaches to such tumors owing to their early appearance and the extended period during which they remain passive, at least fifty-five years having elapsed in one instance between the date of first observation and the onset of inflammatory action.

Although a small they are a peculiarly instructive minority of cases; "for," says Hirschberg, "they show that the disposition to the appearance in later and even advanced age of progressive new growths may be established during fœtal life."

The patient from whom the eye here examined was removed reports himself, six months subsequent to operation, in perfect bodily health, with no irritation of orbital tissues. He is wearing an artificial eye.

The writer desires in closing to express his deep sense of indebtedness to the late Prof. Dr. Otto Becker for uniform kindness and courtesy during a year of study rendered profitable through his advice and direction.

Explanation of the Illustrations.

FIG. 1. Section through anterior segment of inferior half of globe. Magnified $\frac{15}{1}$.

A. Displaced ciliary processes. *B.* Luxated lens, the angle *M.* (about 40°) indicating the relation of its long axis to *L*, a line drawn through antero-posterior diameter of globe. *C.* Shrunken capsule. *D.* A point of rupture in Descemet's membrane *E.* Collection of pigment showing location of remains of iris. *d.* Folds in Descemet's membrane.

FIG. 2. Section of cornea near limbus, showing destruction of Bowman's membrane and separation of epithelial layers. Magnified $\frac{50}{1}$.

A. Bowman's membrane. *a.a.* The same with frayed ends where broken through by sarcoma cells. *B.* Newly formed vessels. *C.* Hypertrophied corneal epithelia near limbus. *D.* Columnar epithelial layer separated from Bowman's membrane.

FIG. 3. Section of cornea with fibrous-tissue invasion and rupture of Descemet's membrane. Magnified $\frac{250}{1}$.

A. Thickened Descemet's membrane. *a.a.* Same ruptured and curled upon itself with splits in its layers. *B*. Altered corneal tissue. *C*. Fibrous inflammatory mass emanating from iris and penetrating cornea. *D*. Vessels of the same.

FIG. 4. Section through posterior segment of globe. Magnified $\frac{100}{1}$.

A. Optic nerve. *B*. Papilla; composed of connective-tissue bundles and inflammatory cells. *C*. Choroidal sheath for the larger arteries and veins; chorio-capillaris. *D*. Bone deposits. *E*. Nerve-fibre layer of retina. *F*. Remains of retina involved in fibrous structure. *G*. Infiltration in sub-vaginal space.

FIG. 5. Section of bone found at posterior pole of the eye. Magnified $\frac{650}{1}$.

A. Marrow-spaces. *B*. Haversian canals with blood-vessels. *C*. Giant-cells—osteoclasts. *D*. Haversian lamellæ. *E*. Lacunæ with contained bone cells and anastomotic canaliculi. *H*. Solution of intervening bony wall between marrow-spaces owing to action of osteoclasts. *P*. Fat-cells in marrow-spaces.

FIG. 6. Showing rupture of lens capsule. Magnified $\frac{650}{1}$.

A. Thickened lens capsule. *B*. Point of separation. *C*. Degenerated cortex of lens into which fibrous tissue and sarcomatous cells have entered.

LITERATURE.

FUCHS, Melanoma iridis. *Archiv für Augenheilkunde*, vol. xi., p. 435.

LEBER, Chorioidealsarkom mit Knochenbildung. *Zehender*, 1883, p. 193.

KNAPP, Melanoma iridis. "Intraocularen Geschwülste," 1868, p. 220.

KNAPP, Primäres Irissarkom "Intraocularen Geschwülste," 1868, p. 175.

HIRSCHBERG, Ein Fall von Sarkoma iridis. *Graef. Archiv*, vol. xiv., 3, p. 289.

GRAEFE, Zusätze über intraoculare Tumoren. *Graefe's Archiv*, vol. xiv., 2, p. 107.

DIXON, Sarcoma of iris and ciliary body. *Medical Times*, 1863, p. 507.

STELLWAG, Sarcoma iridis. *Lehrbuch der Augenheilkunde*, 3 Auf, p. 562.

BERTHOLD, Diffuses zum Theil melanotisches Granulationssarkom des Corpus ciliare und der Iris. *Graef. Archiv*, vol. xv., p. 168.

GRAEFE, Eine melanotische Geschwulst der Iris. *Graef. Archiv*, vol. i., p. 414.

NORDENSEN, Ein Fall von cavernösem Aderhautsarcom mit Knochenschale bei einem elf jährigen Mädchen. *Graef. Archiv*, vol. xxxi., 2, p. 59.

Fig. 4.

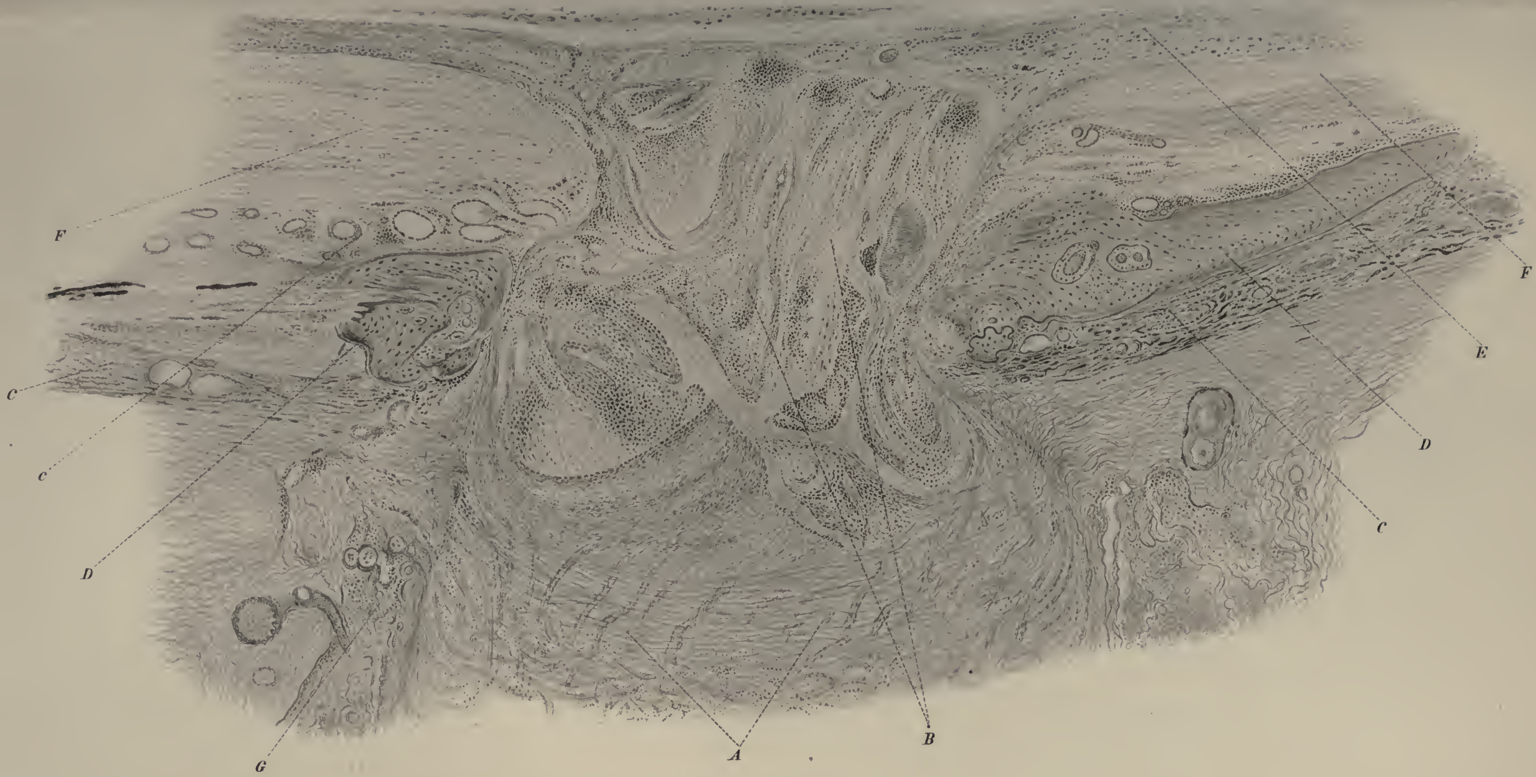

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Fig. 6.

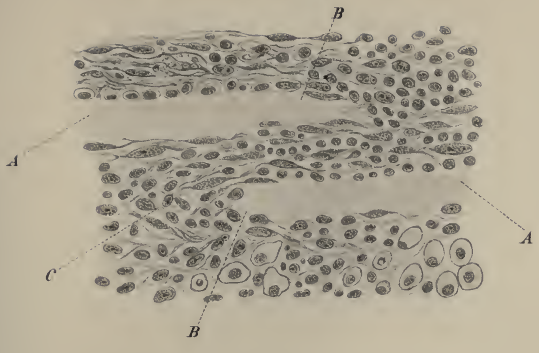


Fig. 2.

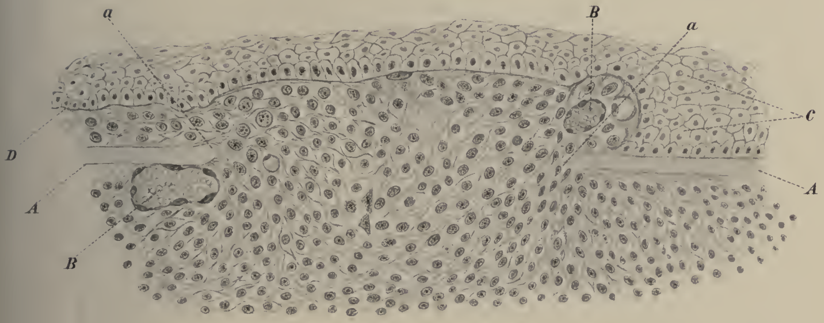
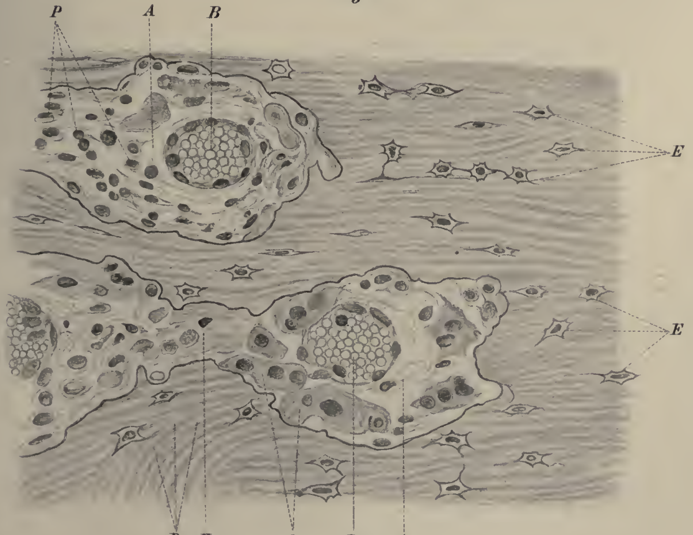


Fig. 5.



J. [illegible] u. H. Petersen.

Lith. Anst. v. C. Kirst, Leipzig.

VOSSIUS, Ein Fall von Tumor Chorioideae. *Graef. Archiv*, vol. xxxi., 1, p. 147.

THALBERG, Zur pathologischen Anatomie des primären Irissarcoms. *Archiv f. Augenheilkunde*, vol. xiii., p. 20.

KNIES, Sechzehn Fälle von Aderhautsarcom. *Archiv für Augenheil.*, vol. vi., p. 175.

KNAPP, Drei erfolgreich operirte Fälle von Sarcom der Iris. *Archiv f. Augenheilk.*, vol. viii., p. 241.

LITTLE, Sarcomatous Tumor of Iris. *Lancet*, 1883, No. 11.

PFLÜGER, Metastatisches Sarcom der Chorioidea. *Archiv für Augenheilk.*, vol. xiv., p. 129.

BINNET. *Quinze vingts*, No. 1, p. 22, 1883.

HIRSCHBERG, Ueber die angeborene Pigmentirung der Sclera und ihre pathogenetische Bedeutung. *Graef. Archiv*, vol. xxix., 1, p. 3.

GRAEFE, Tumoren der Iris. *Graef. Archiv*, vol. vii., 2, p. 36.

ALT, "Compendium der Histologie des Auges," p. 149.

HOSCH, Primäres Sarcom der Iris. *Centralblatt für practische Augenheil.*, 1881, p. 361.

ARNOLD PAGENSTECHER, Beiträge zur pathologischen Anatomie des Auges. *Graef. Archiv*, vol. vii., 1, p. 99.

KNAPP, Ueber Knochenbildung im Auge. *Archiv für Augenheil.*, vol. ii. p. 133.

SCHIES-GEMUSEUS, Beitrag zur Lehre von der Knochenneubildung in der Chorioidea. *Graef. Archiv*, vol. xix., 1, p. 202.

WARREN TAY, Sarcoma of the Iris. *Ophthalmic Hospital Reports*, Bd. v., p. 220.

FUCHS, "Das Sarcom des Uvealtractus." 1882.

KRÜKOW, Fall von primären Sarcom der Iris. *Wjestnik Ophthal.*, iii., 3, p. 179.

WEBSTER, Sarcoma of the Iris. Report American Ophthal. Society, 1885, p. 23.

HASNER V., Melanosarcoma iridis primarium circumscriptum. *Prager med. Wochenschrift*, 1882, p. 61.

HOSCH, Ein Fall von primären Sarcom der Iris. *Correspondenzbl. für Schweizer Aerzte*, No. 8.

PFLÜGER, Zur Kasuistik des Melanosarcoms der Augen. Bericht der Augenklinik f. d. J., 1883, Bern.

KIPP, Ueber einen Fall von primären Irissarcom. *Archiv f. Augenheil.*, vol. v., 1, p. 177.

ROOSA, Sarcoma of the iris. *Trans. Am. Ophthal. Soc.*, 1869.

OSSIFICATION OF CHOROID.

By T. HILLIARD WOOD, M.D., Nashville, Tenn.

Miss C., æt. twenty-five years, suffered with ophthalmia neonatorum. In the right eye the result was to leave the cornea with an opacity occupying its lower half, a partial cataract, with adhesions of iris to the lens. This eye has since retained little more than a perception of light.

In the left eye the result was to at once completely destroy vision, the globe remaining as an atrophied stump not more than two thirds its normal size.

This stump continued tender to the touch, and has always been the seat of recurring attacks of neuralgic pains. These attacks come on every one, two, or three weeks, and last two or three days. The pain was located in left eyeball and corresponding side of the head and face. During these attacks the right eye would become irritable, sensitive to light, and have a free secretion of tears.

On the morning of February 5, 1890, assisted by Dr. Handley, I enucleated the left eye. Upon examination after its removal, the optic nerve was found to be a flaccid cord, about one third its normal size, and of a brown color. The sclerotic had nothing peculiar, and the cornea was replaced by cicatricial tissue. Upon opening the sclerotic, a shell of bone occupied its interior. This shell was about 2 *mm* in thickness in all its parts, and occupied the exact position of the inner layer of the choroid. The degenerated outer layer of the choroid was lying between the sclerotic and bone, but not connected with either. Posteriorly the bony shell presented an opening for the optic nerve. Anteriorly the shell continued to the corneo-scleral junction, where it was loosely connected. The anterior opening in the shell was about the size of a crystalline lens. The outer, convex surface of the shell lay in contact with the outer layer of the choroid and sclerotic, but not connected with either except at the corneo-scleral junction. In the concavity of the shell lay the remains of the retina, with a limpid liquid, representing the aqueous humor. I could detect no vitreous or lens.

The degenerated remains of the iris were present, but no evidence of pupil or ciliary processes.

It will be seen this shell had the exact situation and relations of the capillary layer of the choroid.

Since the enucleation the neuralgic attacks have entirely disappeared; and in the right eye the ability to observe bright objects has markedly improved.

HYPERPLASTIC INFLAMMATIONS OF THE MEMBRANES OF THE EYE.

By Prof. J. SCHÖBL, of Prag.

(With Tables IV.-IX.)

Translated by Wm. F. Smith, M.D., Chicago.

INFLAMMATORY processes of individual membranes of the eye, be they sclera, choroid, or retina, which, after the manner of hyperplastic inflammations of other connective-tissue organs of the body (through extravasation of round cells in masses, their multiplication and development, development of plentiful neoplastic blood-vessels thus forming regular granular tissue), gradually build up tumor-like hyperplasias of the whole or individual portions of the membrane and produce, so to speak, a transition link between inflammatory and neoplastic processes, find as yet so little consideration in ophthalmology that even the term "hyperplastic inflammation" is hardly mentioned. Analogous processes only come into notice here and there, generally, as incidental conditions of atrophied eyeballs.

In this connection an observation of Klebs: "Zur normalen und pathologischen Anatomie des Auges," *Virchow's Archiv*, Bd. xxiv., S. 367, deserves special mention. In a phthisical eyeball on which repeated operations for staphyloma had been made before the enucleation, he remarked an enormous thickening of the retina and choroid, which he designated as the direct result of hyperplastic inflammation of these membranes.

In the following lines I will endeavor to show that hyperplastic inflammations of the membranes of the eye deserve far greater consideration from a clinical standpoint than has been accorded to them so far, that not always do they merely present interesting pathological conditions of phthisis of the globe, but that they occasionally play a more or less important role *intra vitam.*

Before I proceed to the description of particular forms of hyperplastic inflammations in the eye, I will briefly refer to three cases, which more than any others have furnished excellent material for the study of this particular form of inflammation, and which in their clinical course more or less resembled the so-called *pseudo-gliomas.*

CASE 1.—N., Joseph, two years old, was brought to me May 15, 1880, in an incredibly neglected condition. Some eight months before the child had suffered from some exanthematous fever, probably scarlatina. About six months ago it began to complain of pain in the right eye. The mother found the eye reddened and with something white over it and totally blind.

I found the lids of the right eye very much swelled and reddened, the conjunctiva chemotic. The eyeball was enlarged, pressed forward, and immovable. The cornea was flattened, irregularly curved, opaque with smooth surfaces. At its margin were five reddish-white, soft, and very vascular excrescences which had forced their way out from the interior of the eye. The largest was the size of a pea and the others about the size of hemp seeds. I diagnosed *glioma retinæ.* The left eye was entirely normal.

I extirpated the eye with as large a portion of the optic nerve as possible. A clean enucleation was not possible on account of extensive adhesions about the immovable eyeball. The extirpated globe was injected according to my method and the extremely difficult injection was entirely successful. After being hardened in Müller's liquid and picric acid solution, the globe was divided by an incision through the sagittal meridian. The anatomical condition revealed was extraordinarily astonishing (Fig. 1). Of a glioma filling up the interior of the eye no trace was to be found. The cornea, lens, and vitreous body were also missing.

The sclera is much thickened, especially at its posterior portion where its thickness reaches 3½ *mm*, and is permeated throughout with round cells. The space between the capsule of Tenon and the eye is unrecognizable, so that the hypertrophied sclera merges, without boundaries, into the fatty tissues of the orbit.

The uvea, particular sections of which, as ciliary body and iris, are hardly recognized as accumulations of pigment in the interior of the tumor-like masses, is enormously thickened as a whole, so that its volume here and there extends to 5 *mm.* It is composed of granular tissue and large neoplastic blood-vessels almost throughout, and forms the above-mentioned five excrescences which issue from the eyeball in the ciliary region.

I will give its exact histological relations later when considering particular forms of hyperplastic inflammation.

The whole retina, detached and folded funnel-wise, is granulating and forms the anterior wall of the globe, a true retinal staphyloma formed like a staphyloma of the iris. In the clinical examination it was erroneously thought to be a remnant of opaque cornea (Fig. 1 and Fig. 6). The space between the choroid and retina was filled with clotted fibrin masses. The anatomical diagnosis must therefore indicate : *scleritis, choroiditis et retinitis hyperplastica, staphyloma retinæ, granulomata chorioideæ.* The clinical diagnosis by no means coincides with the anatomical. Clinically glioma was diagnosed ; anatomically a hyperplastic inflammation of all the membranes of the eye was found.

This case teaches that hyperplastic inflammations of the membranes of the eye should be considered not only as incidentally interesting conditions of pathological eyeballs, but that from a clinical standpoint also they merit full evaluation.

Should we desire to sketch, with the above-described anatomical data in hand, this pathological process, its course, in all probability, must have been something like the following :

In convalescence after scarlatina the child suffered from an abscess in the cornea accompanied by infiltration of pus, which, on account of miserable hygienic conditions, led to exfoliation of the entire membrane. Expulsion of the lens and vitreous body easily followed, leading to total detachment of the retina, the anterior portion of which obstructed the opening left by loss of the anterior membranes. Under atmospheric exposure and interpenetration of conjunctival secretions the exposed surfaces now acquired the characteristics of granular, connective, and cicatricial tissues—in short, the formation of a retinal staphyloma. So long as the perforation was not hermetically closed by the retinal staphyloma, the choroid remained in an inflammatory condition, which, here too, led to the formation of granular tissues and to excessive hypertrophy of this whole membrane, and to the growth of the tumor-like masses through points at the margin of the staphyloma not yet sufficiently consolidated. Perhaps also portions of the uvea had been prolapsed or entangled between retina and sclera.

By continuity of tissue the sclera also was involved in the inflammatory action. In this manner was formed the pathological picture which, in so many of its features, resembled glioma.

CASE 2.—A four-year-old, extremely wretched and neglected girl was brought to my dispensary June 13, 1876. She had repeatedly suffered from kerato-conjunctivitis phlyctænulosa. Some months before a thorn pierced the sclerotic and was extracted. The eye remained red and was blind, its pupil at times shining yellow-white. The eyeball was congested; T. — ½. Cornea opaque and vascular; iris muddy; pupil partially dilated; no reaction to light; slight to atropine. From the interior of the eye shimmered a yellow-white reflex, proceeding from a mass that seemed to fill more than half the vitreous chamber. Its surface appeared uneven, some portions more reddish than yellow, and it seemed to me I could detect neoplastic blood-vessels here and there. The diagnosis vacillated between glioma retinæ and choroiditis purulenta chronica. The fact that the disease depended on a peculiar wound, as well as the diminished tension, was against glioma. Finally I was inclined to diagnose choroiditis purulenta chronica, as I had seen a few similar pathological conditions after wounds, also one case after an eye had been penetrated by a wooden splinter.

I asked the mother to bring the child every two weeks. She never came at all. Three months later I found the child dying from pneumonia. The eye was diminished in size and tension. Thick opacities hindered any view of its interior. The next day the child died. By means of gentle words and some gold I procured the head of the cadaver.

The entire head was injected through the carotids, and the contents of the orbit together with the lids was exenterated and carefully hardened ; then a sagittal incision was carried through both lids and the globe. The anatomical examination was again a complete surprise to me (Fig. 2). It was indeed a disease of the choroid, as I had anticipated, but a disease of such a character as no one up to the present had ever observed, and such as the most lively phantasy would hardly conjecture. The choroid was vastly thickened, its blood-vessels enormously distended, while in numerous places they broke through the retina with its neoplastic vessels and granular tissue, to build tumor-like growths in the vitreous space, which growths were composed partly of granular tissue, partly of epithelioid cells, and partly of cells that were not to be distinguished morphologically from true epithelium.

The retina also is very much thickened, the granular layer of which, twisted in numerous zig-zag folds, is the only recognizable portion ; of the rest, both inner and outer layers have been transformed into connective tissue. As before mentioned, the choroid had grown through the retina at numerous places. The lens is opaque and unevenly shrunken. In the cornea a few deep blood-vessels are found, the sclera thickened by shrinkage, its fibres bent in zig-zag folds.

The anatomical diagnosis therefore must read : *atrophia bulbi progrediens, retinitis et choroiditis, hyperplastica, cataracta secundaria (siliquata).*

Case 3.—A nine-year-old, healthy-looking boy was brought to my dispensary in May, 1881. Some months before he had received a blow in the left eye from the horn of an ox. The eye was only reddened, but there was continuous pain, photophobia, tears, and gradual loss of vision. The globe of the left eye, with its appendages, was normal as to form, position, movement, and tension, but the ciliary veins were somewhat distended and tortuous, the conjunctival ring deeply injected, and the region of the ciliary body sensitive. The cornea was seamed and slightly smoky, but transparent ; the chamber rather shallow ; iris discolored, its structure not clear ; the pupil narrow and immovable ; lens transparent. From the interior of the eye shimmers a white-yellow reflex, which under proper illumination is seen to come from masses close behind the lens. The patient complains of severe pain that increases during the night.

My diagnosis was iridochoroiditis traumatica, considering the white-yellow masses as the product of cyclitis deposited in the anterior region of the vitreous.

In this case also one might easily suspect glioma, as the counterfeit of an amaurotic cat's eye was exquisitely given ; the tension was rather increased than diminished, while the suspected choroiditis did not contradict the presence of a tumor. Enucleation refused. The right eye was entirely normal.

In July of the same year the patient returned, desiring immediate removal of the blind eye on account of severe continuous pain. I found the globe smaller, flattened, sensitive to pressure ; T. — .5, movement retained ; the chamber very narrow, structure of the iris unrecognizable, and the pupil obstructed by inflammatory products.

In accordance with these conditions I now modified my former diagnosis into : *atrophia bulbi progrediens ex iridochoroiditide traumatica, keratitis interstitialis inducta, occlusio pupillæ.*

The globe was enucleated and the patient discharged on the fourth day.

The anatomical conditions discovered by a meridional incision through the globe, hardened in the usual manner, were again astonishing (Fig. 3). The clinical diagnosis, iridochoroiditis, was certainly confirmed.

The choroid is thickened and infiltrated throughout with round cells, which, with pus and fibrinous clots, form layers on both its surfaces. The iris and ciliary body are also infiltrated with round cells. The posterior chamber and pupil are filled with granulations and young connective tissue. The ciliary muscle is atrophied. The cornea contains many groups of round cells and deep blood-vessels. The endothelium of the anterior capsule of the lens is hypertrophied and covered with quite a thick layer of neoplastic connective tissue. In the cortical portions of the lens the fibres are broken, twisted, and infiltrated with myelin molecules and detritus masses.

The most evident and important changes, however, are shown by the retina, which as a sort of compact tumor nearly fills the vitreous chamber, while the vitreous itself, as a relatively small and shrunken body in the form of a double cone, adheres to the posterior pole of the lens.

The whole transformed retina consists almost entirely of granulations and fresh connective tissue, while of the original retinal elements only slight traces can be recognized.

The anatomical diagnosis therefore must stand : *atrophia bulbi progrediens ex iridochoroiditide traumatica*, *retinitis hyperplastica*, *keratitis interstitialis inducta*, *occlusio pupillæ et synechia iridis posterior membranacea totalis*, *phacohymenitis*, *cataracta corticalis*.

In this case also the products of hyperplastic inflammation, revealed by the shimmering reflex or amaurotic cat's eye, came under observation at the first clinical examination and formed an important factor in the diagnosis, in which it may eventually play the deciding rôle.

In addition to these three somewhat similar cases, many specimens from my rich collection present changes here and there which must be referred to hyperplastic processes in the different membranes of the eye. Since they are, collectively, less surprising than those above described, and do not present any decided deviations ; since, too, the history of many is lacking, I will pass over them here and proceed to the description of particular forms of hyperplastic inflammations.

A.—*Choroiditis Hyperplastica.*

Choroiditis hyperplastica occurs in the first and second of the above reported cases. Since each of the two possesses its own special and interesting peculiarities, we must consider them separately.

In Case I we must again distinguish between the intraocular part of the choroid and those five extruded granulomas.

1. *The intraocular part of the choroid* (Fig. 1). The entire choroid is transformed into a tumor-like mass that lines the interior of the globe to a depth of from 1½ to 5 *mm.* Externally it is bounded everywhere by the sclera, though the limits in many places are not well marked. The inner surface of the choroidal mass is coated throughout with retinal pigment epithelium, forming a more or less irregular layer. Anteriorly the choroid is directly merged into the

projecting granulomas, at the base of which twisted masses of pigment are seen, which are doubtless to be considered as the rudiments of the iris and ciliary body.

It must be premised that almost nothing remains of the normal, histological structure of the choroid, the entire membrane being represented by a tumor-like mass resembling granular tissue permeated with numerous neoplastic blood-vessels. The whole mass is made up of round cells, intercellular substance, young connective tissue, and blood-vessels.

The round cells, with the intercellular substance, make up the greater portion of this tumor-like mass. Probably all, or at least the most of these cells were irrupted from the blood-vessels. At any rate, round cells can be seen just emerging from the vessels in numberless places, and the greatest accumulations are found in closest proximity to the blood-vessels. These accumulations are thickest on the inner surface, just where the mass was in constant process of growth, as is evident from the new formation of blood-vessels at these points.

The intercellular substance is rather scanty. It comes into view, by aid of strong immersion systems, at one time as a reticular, again as a granular, structure; however, I believe this depends on the effect of the hardening fluid, and that during life it might have exhibited a semi-fluid, protoplasmic consistence.

The blood-vessels are extremely numerous and largely developed. The largest vessels are found in the external part of the growth, near the sclera, and doubtless represent the colossally enlarged vessels of the *stratum vasculosum Halleri*. From here the neoplastic blood-vessels pass centripetally to the inner surface; they are, as a whole, thin-walled and form no real capillary network, the finer arterial twigs merging immediately into venous twigs. Near the inner growing surface of the mass numerous blind outgrowths from the blood-vessels are found, which doubtless represent the formation of new vascular branches.

Connective tissue is found as a more or less continuous layer on the periphery of the mass near the sclera—that is to say, in the oldest parts of these tissues. Moreover, it accompanies the largest neoplastic blood-vessels in more or less powerful bands. The gradual development of round cells into young connective tissue can be observed everywhere.

The entire intra-ocular portion of the choroid comports itself like granular tissue of luxuriant growth, or like an atypically growing tumor. Near the inner surface new round cells constantly emerge from the blood-vessels and proliferate, and the blood-vessels send out new branches, and it is here that the growth of the mass continues, while in the older portion layers of connective tissue are constantly formed by the gradual transformation and augmentation of round cells.

Whether the stroma cells of the choroid have taken any part in the process of growth cannot be determined on account of the advanced stage of development; in any event they could only have played an inferior rôle, since the round cells, emerging from the blood-vessels, did the lion's share of the work.

2. *The extraocular granulomas of the uvea* (Fig. 1 and Fig. 4), taken together, have the same structure as the intraocular masses of the choroid, consisting of round cells, intercellular substance, blood-vessels, and connective tissue, but differ in that their surfaces are covered with a layer of epithelioid cells, and in that their round cells, collectively, are found in a state of rank proliferation. Probably their exposure to the direct influence of the atmosphere caused their growth to be much more luxuriant than that of the intraocular masses.

As to the *epithelioid covering* of these small tumors, it did not develop, as one might easily suppose, from pre-existing epithelium of the neighboring tissues, but the cellular layer here referred to was formed, as one might directly observe, by gradual, progressive transformation of the round cells.

At the boundary of the neoplastic blood-vessels, near the periphery, round cells are seen which have a broad border; further some with the border broader and still broader, till one comes to epithelioid cells, and finally to such cells as cannot be distinguished, morphologically at least, from true epithelium ; and at all points so numerous and so gradual are the transitions between the extremes of cell-formation that it is impossible to doubt for an instant the transformation of round cells into those of epithelium form. These relative conditions are presented in Fig. 4.

The round cells in the region of the blood-vessels, on the other hand, almost all contain 2–3 or more, up to 6, nuclei, and I cannot believe that exactly here, where the process of growth advances with the greatest luxuriance, almost all of these cells should be destined to destruction, the less so since near the periphery may be observed a continued development of epithelioid cells, and near the base, into something similar to fibro-plast and young connective tissue. Both cells and nuclei have a perfectly healthy and regular appearance, so that I cannot but consider these multiple nuclei as the direct product of nuclear division, and the cells of this portion as in a state of exceedingly luxuriant propagation (see Fig. 4). As to the repartition and formation of fresh connective tissue in the granulomas, it coincides perfectly with that which occurs in the intraocular, uveal masses.

3. The hyperplastic choroiditis in Case 2. (Fig. 2 and Fig. 5) presents many and very important deviations from that form just described.

The original, colossally thickened choroid, permeated with round cells and with pigment, is still partially recognizable, at least the *stratum vasculosum Halleri* and *supra-chorioidea* may be distinctly distinguished. The vessels of the *stratum Halleri* have prodigious dimensions, and from them arise large neoplastic blood-vessels which, with their accompanying granular tissues, break through the thickened retina and, passing centripetally, reach the vitreous chamber. This chamber is now filled up with a tumor-like mass, which in the peripheral portions, so far as the neoplastic blood-vessels have reached through the retina, consists of granular tissue as we have described it in the previous case, whilst in the centre an enormous bed of cells is found which cannot be distinguished morphologically from epithelium.

No trace of the vitreous body is found. On closer histological examination of these masses grown into the vitreous chamber through numerous perforations in the retina, we find an intermediate zone corresponding (in position) to the finer branches of the neoplastic blood-vessels which, in addition to these vessels, consists only of round cells and intercellular substance. Here the round cells are uncommonly thick, and thickest along the walls of the vessels. Here may be observed round cells in great numbers just emerging from the walls of the vessels, in many of which cells direct nuclear division is in evidence.

A progressive development of the round cells may be seen from this intermediate zone in two opposite directions. Near the centre of the vitreous chamber one sees how they change into epithelioid, and then into complete epithelial cells. Even near the limits of the vessels one sees a few round cells with broad protoplasm, and further toward the centre continually wider and wider borders till perfect epithelioid cells are reached. All possible transition forms of both kinds of cells, side by side, can be perfectly seen here too (Fig. 5), as before described at the periphery of the granulomas.

Though the transition of round cells into epithelium might have been doubted as to the uveal granulomas, here in the case under consideration, all possible doubt must be removed.

The tumor-like growth of the choroid which fills the vitreous chamber is more or less like a ball in form (see Fig. 2)

The kernel of the ball is a great bed of these epithelioid cells free from blood-vessels, the cells being separated from each other only by scanty intercellular substance. The peripheral substance of the ball, however, is composed entirely of round cells and blood-vessels. The epithelial cells, therefore, appear to be surrounded by a thick layer of round cells like a mantle, and the existence of such cells in the midst of the vitreous chamber in the centre of a ball of granular tissue under any other methods of growth or being is inconceivable. Now when at all points between the zone of round cells and the central epithelial bed are seen numberless cells in all possible stages of transformation from the one to the other variety, every logical conclusion must be rejected if the genetic relationship between these two forms of cells is to be denied. On the other hand near the outer surface of the retina, and at the points of eruption through that membrane, the round cells gradually develop into connective tissue in a manner similar to that we have mentioned in the previous case.

This histological condition is certainly astonishing and extremely interesting in the truest sense, and in many connections of eminent importance also.

No similar case of numerous perforations of the retina by the growth of inflammatory products has ever been observed so far as I know; even malignant tumors do not grow through the retina so freely.

Further such an enormous development of epithelioid cells in the midst of an inflammatory growth of the choroid appears to be something enigmatical, if not quite without parallel, for Fedor Krause has observed something distinctly similar, to which I shall refer more in detail later.

What would have been the further development of that enormous central bed of epithelioid cells had not the process of growth been arrested by its death, I cannot conjecture.

The appearance of epithelioid cells isolated in the midst of granular tissue is very important as an irrefutable proof, as I believe, of the possibility of the progressive development of round cells into cells like epithelium.

The entire ophthalmological literature relative to diseases of the choroid furnishes only two cases which in some measure at least resemble those above described.

The first of these is extremely interesting, and was reported by Fedor Krause from the clinic of Prof. Hirschberg: "Beitraege zur Pathologie der sympathischen Augenentzündung," *Archiv für Augenheilkunde*, Bd. x., S. 629. It concerns the eye of a girl which was blinded by a cut made by a piece of glass, the injury being followed by sympathetic inflammation of the second eye.

In the enucleated eye Krause found the entire choroid so thickened that it was about the depth of the normal sclera. The hæmatoxylin colored sections seemed flecked dark and pale; microscopically the dark spots proved to be collections of round cells, the pale spots beds of epithelioid cells and cells that differed in no respect from epithelium. Besides these, giant cells were also found.

H. Pagenstecher published the second case: "Zur Casuistik der Augenverletzungen," *Archiv für Augenheilkunde*, Bd. viii., S. 65. This was the case of a man who had been struck in the eye by a cow in such a manner that the optic nerve was torn from the globe. On anatomical examination Pagenstecher found the entire choroid thickened. Histologically this choroid was of alveolar structure throughout, consisting only of connective tissue, its cavity being lined with endothelium.

Most probably the two cases of Schiess belong to this category also: "Fibröse Degeneration und Fibrom der Chorioidea," *Virchow's Archiv*, Bd. xlvi., S. 53.

The first was an atrophied globe without clinical data, in which the choroid was thickened and transformed into connective tissue. The second concerned the eye of a woman which had suffered a severe wound some six months before. In the enucleated eye a white, membranous mass was found over the choroid, which Schiess held to be a fibroma chorioideæ. Without doubt Falkson's case belonged to the same class: "Ein intraocularer Tumor," *Zehender's klin. Monatsbl.*, 1879, S. 193.

In an eye that had been enucleated after years of painful iridochoroiditis Falkson found an enormous mass of connective tissue in place of choroid and retina.

Possibly the case of Michel belongs here too: "Ueber Geschwulst des Uvealtractus," *Graefe's Archiv*, Bd. xxiv., 1, S. 131, which describes a fibroma in one eye and a fibrochondroma in the other eye of the same person.

It would carry us too far to make special mention of all these cases here, an uncommon number of which are found recorded in the literature of the subject.

B.—*Retinitis Hyperplastica*

is found excellently developed in Case 3, less so in Case 2, and only partially so in Case 1.

In Case 3 the retina, grown into a tumor-like mass, fills, as we have before stated, almost the entire vitreous chamber (see Fig. 3). Of the vitreous body only a small residuum remains.

The individual layers of the retina are not recognizable and there are only two large and two small islets, quite isolated, surrounded by granular and connective tissue, in which may be distinguished manifold, twisted, zig-zag rows of granules and radiating streaks which must be considered as residua of the original granular layers.

All the rest of the tumor-like mass consists of granular tissue and fully formed connective tissue, the distribution of these two kinds of tissue being of such a nature that the granular occupies the innermost part which bounds the remnants of the vitreous body and where, without doubt, the mass was in process of growth when the enucleation suddenly put a stop to it. The fully formed connective tissue, on the contrary, is found in the external portions of the mass. Between the two are found numerous transition forms, the granular tissue consisting of round cells, intercellular substance, and blood-vessels, just as in the above-described case of choroiditis hyperplastica, only the former are more scantily distributed and are of weaker development than in the uveal forms. The largest accumulations of round cells are found, here too, nearest the vessels.

The presence, everywhere to be observed, of all possible transition forms between round cells and connective tissue cells is very interesting and important (see Fig. 6).

Of the round cells, thickly accumulated near the blood-vessels, some are found the nuclei of which are oval, then some the protoplasm of which is lengthened either in one direction or in two opposite directions. Further, cells are found the nuclei of which seem graded in length, while their protoplasm is drawn out in long and longer points, so that they acquire an exquisite, spindle-like form. Still further, these spindle-cells are found continually increasing in length, the protoplasm being drawn out in both directions. Finally, cells are found with nuclei cylindric in form, with the protoplasm in long bands, so forming bundles of such cells, and constituting completely formed, young connective tissue. At all stages, from the genuine round cells to the very long, ribbon-like, completed connective-tissue cells, some may be seen the nuclei of which are in process of division, or in which two or three isolated cells may be seen in a mass of common protoplasm.

This condition is presented exactly drawn from nature as seen under a very high power, Seibert, homogeneous immersion $\frac{1}{20}$ (see Fig. 6). These conditions, namely, the accumulation of round cells extruded from and immediately surrounding the blood-vessels, then all possible and imaginable transition forms between these and connective-tissue cells, till, after a certain space, fully developed connective-tissue cells in bundles and beds are found, I believe justify me in the conclusion that development and transformation of round cells have taken place here, also throughout the entire mass of the tumor, and that all the connective tissue here found, in all probability, has been formed in like manner.

1. Every connective-tissue fibre in the case before us corresponds to a connective-tissue cell. The connective-tissue fibres are the outgrowth of the protoplasm of the respective cells into very long and relatively strong bands (see Fig. 6). The development of connective tissue from connective-tissue cells can be directly observed in this case in exactly the same manner it was observed by Valentin in his time (Artikel "Gewebe" in R. Wagner's "Handwörterbuch der Physiologie," 1842, Bd. i., S. 670).

2. The retinitis hyperplastica of Case 2 requires only slight consideration. The entire retina is very greatly thickened, to 2 *mm* and over, and has been broken through in many places, as before remarked. Only the granular layers are recognized as more or less thick, zig-zag lines. Internally and externally is found a layer of connective tissue which in all probability originated in the same manner as we have fully described it in the former case. Only it appears that here the process is more decided and the connective tissue had been formed for a longer time, because only a little fresh granular tissue is present here and there and the transition forms between round- and connective-tissue cells are

scantily found. It appears, therefore, that in this instance the process was more sluggish, whilst in the choroid the growth was an exceedingly luxuriant one.

3. The staphyloma of the retina in Case I (Fig. I and Fig. 7). On the posterior surface of the staphyloma, where, in staphyloma of the iris we usually find remnants of pigment, here we find the twisted granular layers of the retina. Above the granular layer we see rather numerous blood-vessels, in the neighborhood of which we continually find large accumulations of round cells, then follows a layer of round cells and intercellular substance, in which we recognize numerous transition forms from round cells to connective-tissue cells as well as to epithelioid cells, and finally follows a dense layer of connective tissue covered with epithelioid cells. Here also, I have not the slightest doubt, this dense connective-tissue layer originated through the transformation of the round cells extruded from the blood-vessels of the retina.

In all these cases of retinitis hyperplastica we find, as before in the choroid, at first a luxuriant growth of granular tissue, leading later to the formation of connective tissue.

There is hardly any doubt that all the round cells that make up the respective granular tissue were extruded from the blood-vessels, or were derived therefrom. Still less doubtful is the derivation of the collective connective tissues from these same round cells.

Through this observation the existence of true connective tissue in the retina finds its natural explanation and loses all its strangeness; on the other hand, the appearance of connective tissue in the same cannot be considered else than a proof of the pure connective-tissue nature of the supporting tissues of the retina, as was maintained by Klebs (l. c.) in his time.

In searching literature for analogous cases, two reported by Raab are specially worthy of notice. In both cases the eye was enucleated on account of a clinically diagnosed glioma, and in both the anatomical examination showed that the detached retina had been transformed into a tumor-like mass of connective tissue. It cannot be doubted that this condition was preceded by a process such as I have described as retinitis hyperplastica.

I am entirely convinced that in all cases where a fibrous degeneration of the retina is found as an anatomical condition this process of retinitis hyperplastica, as I conceive it, has preceded it, and that connective tissue in the retina cannot be formed in any other manner. To mention all these numerous cases in detail would carry us too far.

The form of disease instanced by Manz as retinitis proliferans (*Graefe's Archiv*, Bd. xxvi., 2, S. 55), also appears to me a nearly related process.

C.—*Scleritis Hyperplastica*

is only found in Case 1 (see Fig. 1 and Fig. 8). The entire sclera is very much thickened, especially in its posterior portion where it is 2½ *mm* and over. It is extremely vascular and infiltrated throughout with round cells, the greatest accumulations of which are constantly to be found in the immediate neighborhood of the blood-vessels. Upon closely examining fine sections with a strong objective, one sees again numberless transition forms from round cells to connective-tissue cells, so that there can be no doubt the entire scleral membrane was undergoing continuous new formation of connective-tissue fibres. Here, too, it is a question of an analogous process, the exudation of great quantities of round cells from the vessels and the transformation of round cells into young connective tissue, only in this case the process is more diffused throughout the entire membrane. The relative bearing of the outer, posterior portion of the sclera is very interesting, where, on account of the surrounding fatty tissues of the orbit the boundaries are not distinct and through constant apposition of newly formed connective tissue it grows like a tumor (see Fig. 8).

The several blood-vessels of the fatty tissues are seen to be surrounded by round cells, which, with intercellular substance, have distributed themselves among the fat cells in various sized groupings.

In many places lengthened, spindle-formed cells may be observed, which, since all possible transition forms may also be found, must have developed from round cells. Here and there individual fibrils are seen in the intermediate substance, so that it appears exactly as if here the formation of young connective tissue would follow in the manner first assumed by Henle.

In other places between the fat cells islets of completely formed connective tissue are found, which occasionally unite with the outer border of the scleral margin, so that it may almost be taken for granted that these islets of connective tissue in process of formation round the blood-vessels, being massed together, are gradually welded with the sclera, and so the extraordinary thickening of the latter takes place in two ways: through new formation of fresh connective tissue from the round cells permeating the sclera itself, as well as through the connective-tissue formation in the surrounding fatty tissues and the subsequent welding of the same with the growing sclera.

We search literature in vain for analogous cases, unless perhaps two cases described as fibroma of the sclera may be reckoned herewith, which may well be considered as the termination of an expired scleritis hyperplastica; the one from Narkiewicz Jodko, *Gazeta lekarska*, 1874, No. 51: "Tumor Scleræ et chorioideæ." After scleritis a staphyloma-like protuberance of the sclera developed at the corneal margin and extended nearly to the equator, which, at the anatomical examination of the globe, enucleated for the relief of extreme pain, proved to be a mass of connective tissue. At the same time fibrous degeneration of the choroid was found in a corresponding position.

The second is the well-known case from Sæmisch: "Fibrom der Sclera," *Archiv für Augenheilkunde*, Bd. ii., S. 115.

In a globe enucleated for a diagnosed glioma, was found a diffused thickening in the posterior portion of the sclera, 10 *mm* long, 8 *mm* broad and 5 *mm* thick. In a corresponding position was found fibrous degeneration and thickening of the choroid and retina.

In both cases the tumor was like a thickening of the sclera without well defined boundaries; in both cases fibrous degeneration of the inner membranes of the eye was found, which facts very much favor the assumption of an original circumscribed, hyperplastic inflammation.

Taking together all I have observed in these three typical cases, and reviewing those reported in literature, which indicate the final result of similar processes, and in all probability should be reckoned herewith, we may state the following *preliminary deductions concerning hyperplastic inflammations of the membranes of the eye.*

1. Etiologically hyperplastic inflammation, in the majority of cases, seems to be the result of injury, more or less severe, to the eyeball, but may also result from long-continued idiopathic, purulent inflammation following diverse causes of irritation.

It occurs more often in badly nourished, reduced individuals, and attains the most remarkable development where a certain greater vulnerability, and capability of reaction to diverse irritations, on the part of the membranes seem to exist. Youth and especially childhood seem to predispose.

2. On those occasions when it does come under observation in the clinic, it appears under the likeness of an intraocular or an extraocular tumor, mostly that of glioma, and most frequently presents the symptoms of the amaurotic cat's eye; it may, however, simulate a tumor already extruded from the interior of the globe, or an irido-choroiditis with apparently enormous cyclitic products in the vitreous chamber.

3. In diagnosis it might more often be mistaken for a tumor; in differential relations a previous history of wound or of a long-continued inflammatory process should have weight.

4. In anatomical connections hyperplastic inflammations of the membranes of the eye are very similar to the granulation process which leads to hyperplasia, and form, so to speak, a natural transition from inflammatory to neoplastic processes.

In general these growths are accompanied by enormous exudation from the blood-vessels, and augmentation of round cells and intercellular substance and new formation of the former, and so to the formation of granular tissue, then to the development of round cells into connective tissue, exceptionally also to cells which cannot be distinguished, morphologically, from epithelium.

5. Therapeutically considered, where malignant tumor cannot be excluded with certainty, where the pain may be extreme, or as a preventive when the possibility of sympathetic disease is present, enucleation should be practised as promptly as possible.

In closing I may remark that it should be of general interest to follow the observations here made in the three cases presented concerning the development of connective tissue out of round cells extruded from the vessels, either if this process is taken to follow the paradigm Valentin had formulated, to wit, that every connective-tissue fibre corresponds to a cell, or that of Henle by which the connective tissue-fibres were supposed to be developed from the intercellular substance, the connective-tissue cells from the round cells, then, moreover, the epithelial cells (morphologically considered) from just such cells.

Explanation of the Cuts.

FIG. 1.—Meridional section through an eyeball with choroiditis and scleritis hyperplastica and staphyloma retinæ, described under Sec. 1, low power. The details are so clear that a special explanation seems unnecessary. I will only remark that in this figure, as well as in Fig. 2 and Fig. 3, the histological elements, in relation to the low power used for the entire preparation, are too large, necessarily so to insure clearness. Otherwise the figure is drawn exactly after nature.

FIG. 2.—Meridional section of Case 2. Choroiditis and retinitis hyperplastica. The growing choroid breaks through the retina in many places. The entire vitreous chamber is filled with epithelioid cells and the lens is shrunken.

FIG. 3.—Meridional section of Case 3. Retinitis hyperplastica, etc. Almost the entire vitreous chamber is filled up by the enormous hypertrophied retina. Of the vitreous only a small dumb-bell shaped residuum is left.

FIG. 4.—Section through the peripheral portion of one of the

uveal granulomas of Case 1 under high power, Seibert, water-immersion, No. VII., Ocular 1. It shows the terminal branches of two neoplastic blood-vessels, one artery and one vein, surrounded by round cells, the nuclei of which seem in a state of active subdivision. Above the blood-vessels are seen numerous transition forms between round cells and epithelioid cells, a continuous layer of which constitutes the upper margin of the preparation.

FIG. 5.—Section through choroid and retina Case 2, rather high power, Seibert, Obj. VI., Ocular 2. Bordering on the sclera below is the wide-meshed supra-chorioidea, and then come the enormous blood-vessels of the stratum Halleri, then the perforated places through the greatly thickened retina, through which the neoplastic blood-vessels arising from the vessels of the stratum Halleri force their way into the vitreous chamber. Between the neoplastic blood-vessels large collections of round cells are seen. Further upwards numberless transition forms between round cells and epithelioid cells are found, and beyond the blood-vessels, finally, a continuous layer of those cells which cannot be distinguished morphologically from epithelium.

FIG. 6.—A small portion of the growing retina of Case 3, under very high power, Seibert, homogeneous immersion $\frac{1}{20}$. A small blood-vessel is seen to the left surrounded by numerous round cells, then further to the right all conceivable transition forms from round cells to connective-tissue cells, and finally, to the right, perfectly formed connective-tissue cells after the paradigm Valentin had formulated.

FIG. 7.—A section through a portion of the staphyloma retinæ of Case 1, under a very high power, Seibert, water immersion No. VII., Ocular 1. Beneath the twisted granular layers is seen clotted fibrin with filaments of fibrin. Above are the blood-vessels surrounded by round cells; then granular tissue with transition forms from round cells to connective tissue, then a heavy layer of connective tissue, and finally a mass of epithelioid cells.

FIG. 8.—A section through a portion of the posterior border of the sclera near the surrounding fatty tissues from Case 1, under rather high power, Seibert, Obj. VI., Ocular 1. The sclera is shown to be infiltrated with round cells. Among the fat cells are islets of newly formed connective tissue which are partly united to the sclera. Beneath are transition forms between round cells and connective tissue.

Fig. 1.

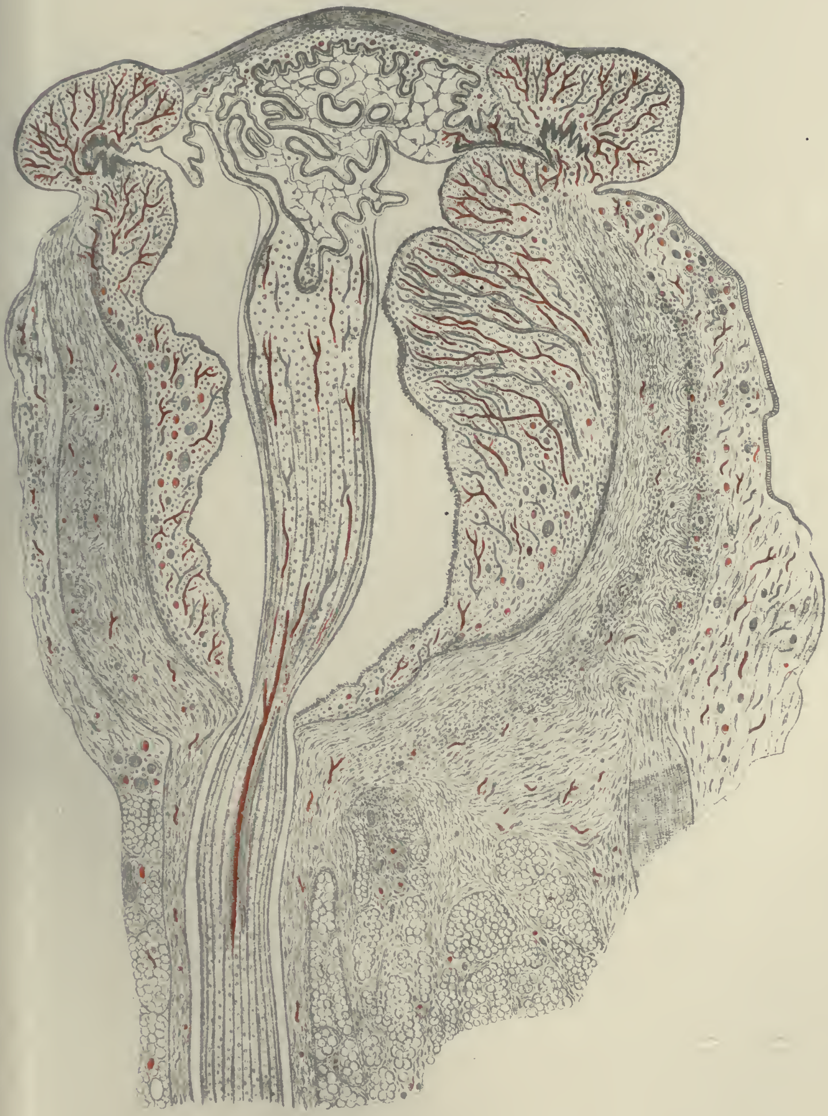


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Fig. 2.

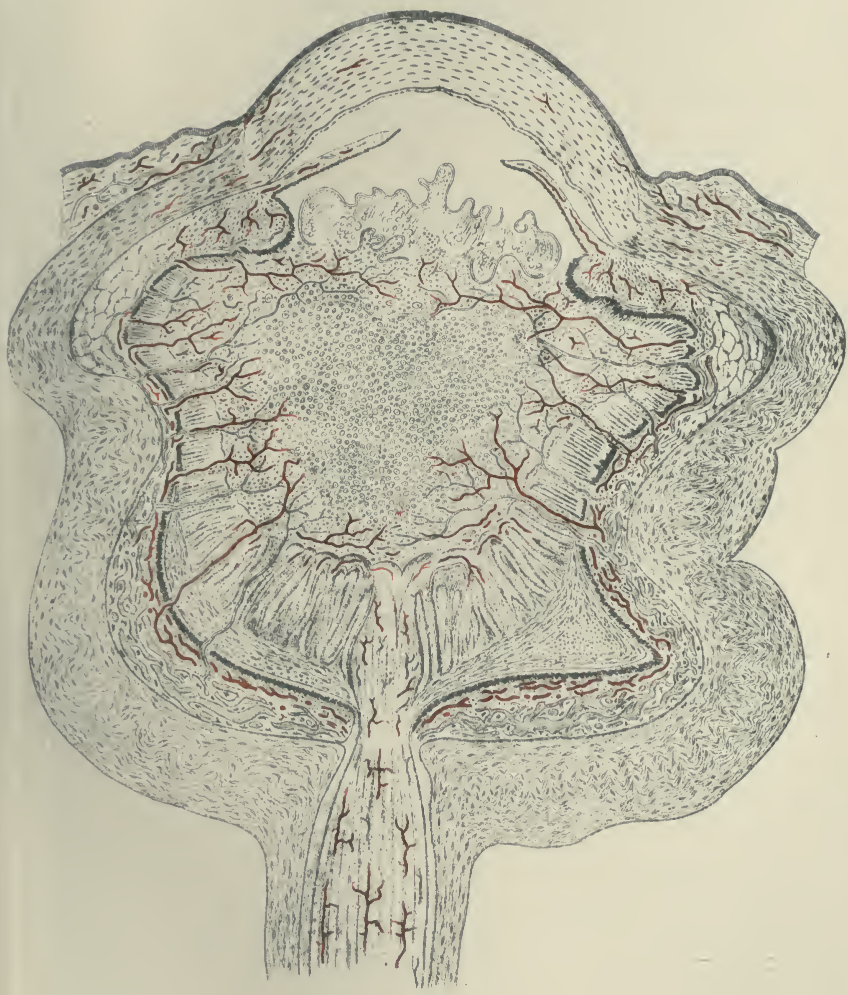

Fig. 3.

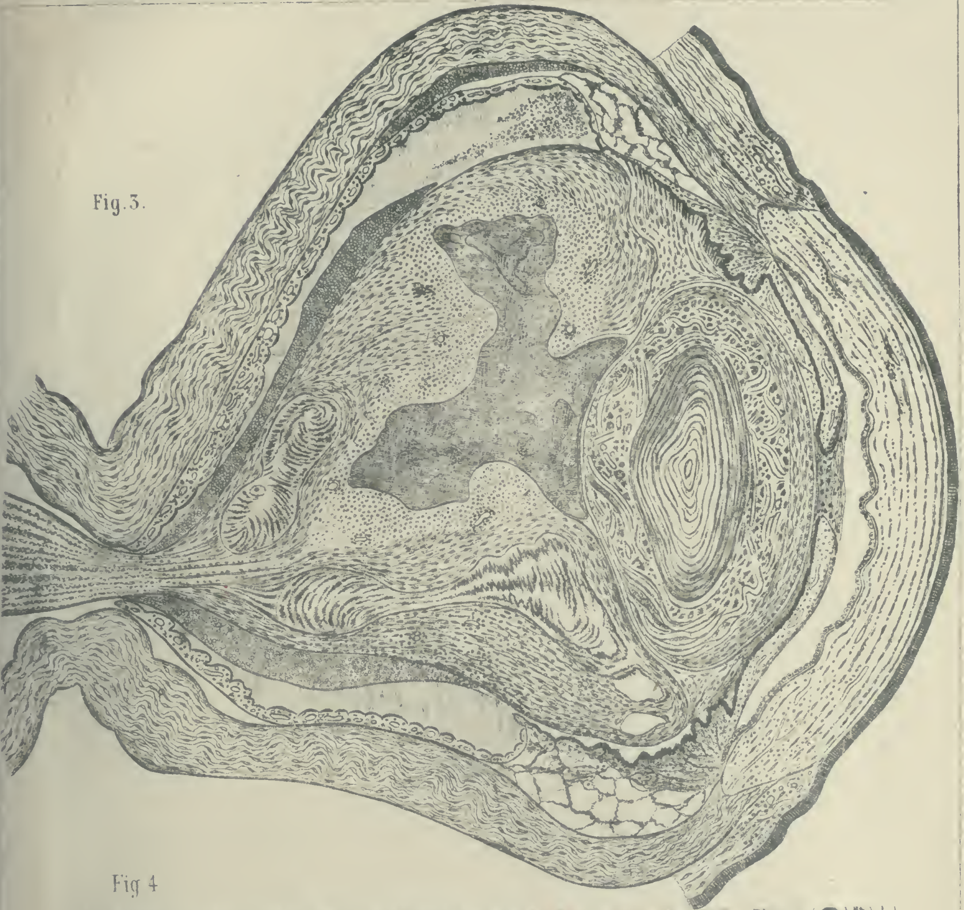

Fig 4

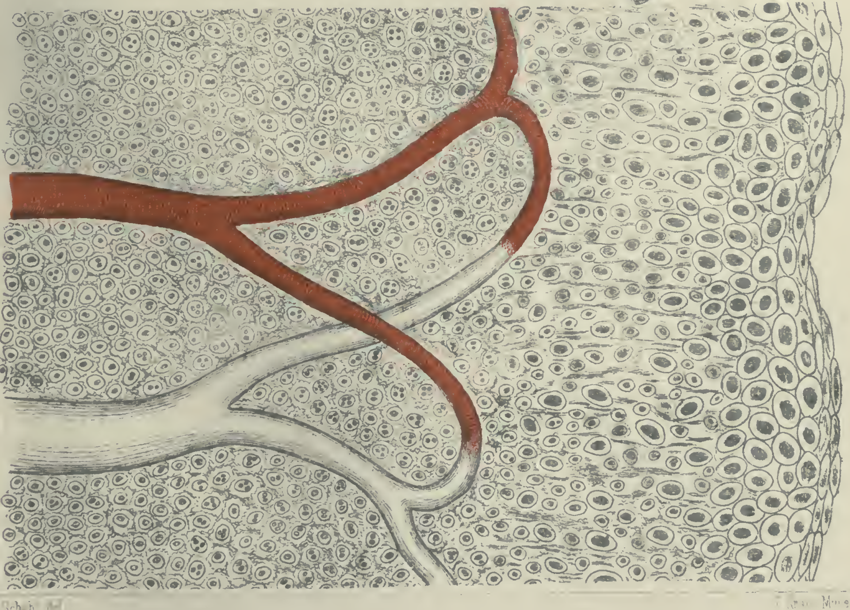

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Fig. 5

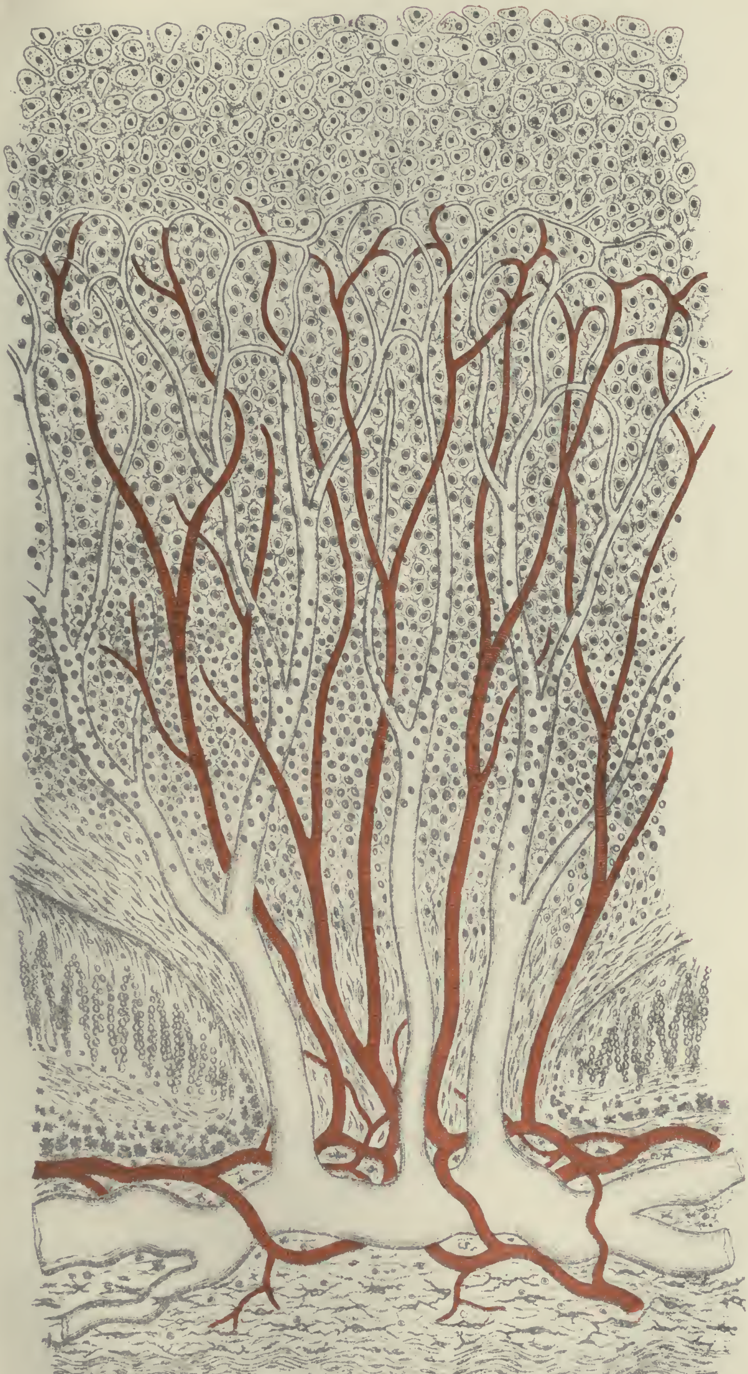

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Fig 6

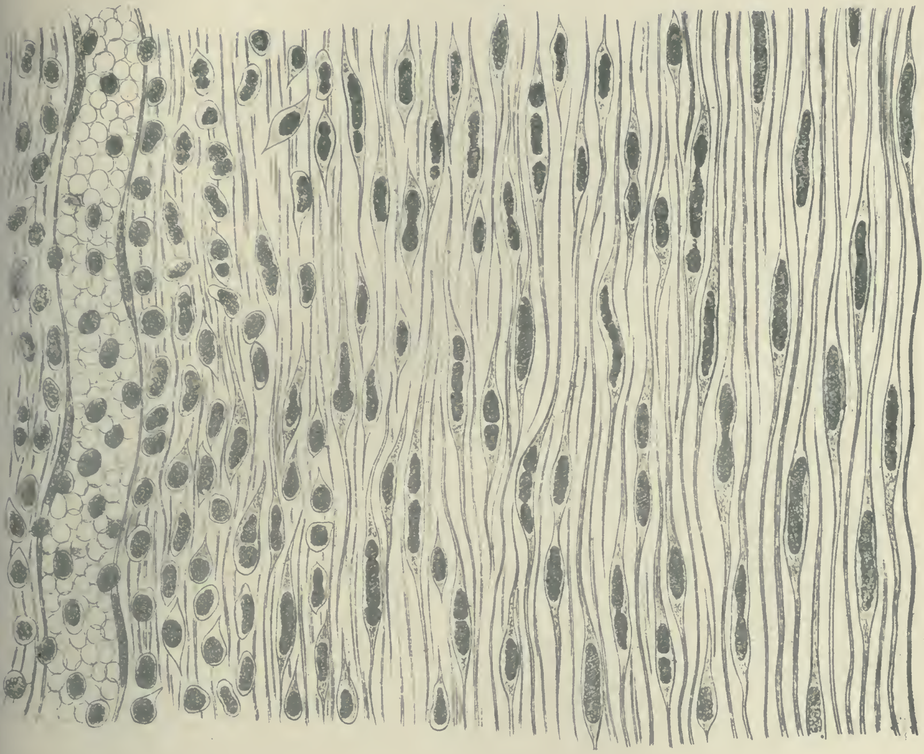

Fig. 7.

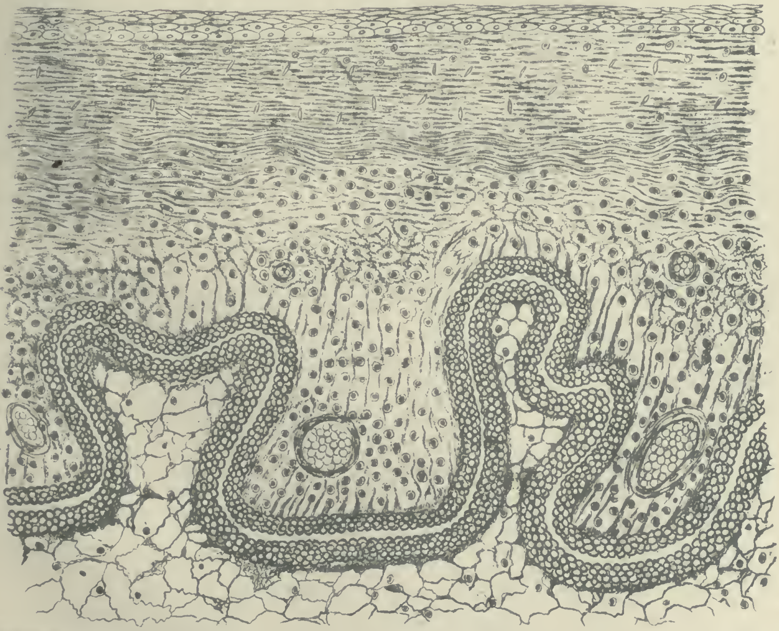

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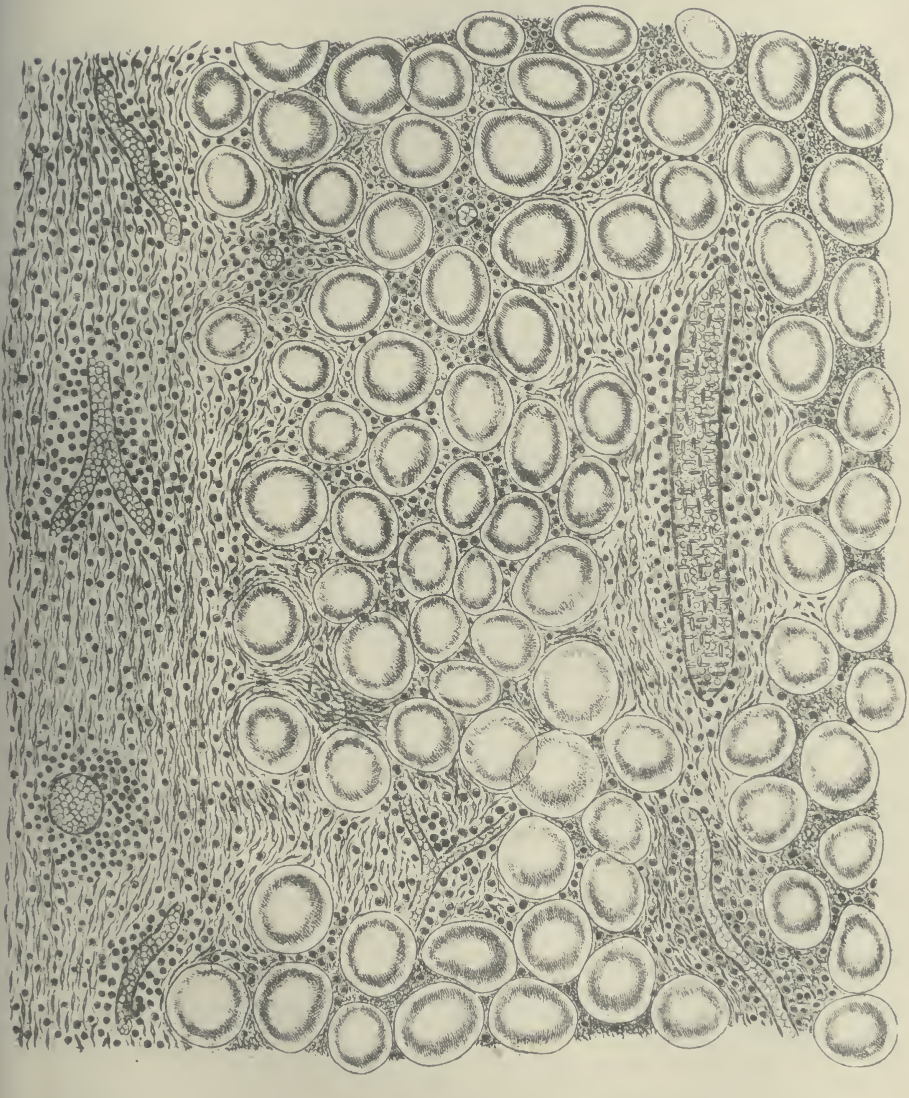
Fig. 8.



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DISTICHIASIS AND TRICHIASIS OPERATIONS.

By Dr. E. JAESCHE, of Dorpat.

(With six illustrations in the text.)

Translated by Dr. H. A. B. Macauley, of New York.

IT is an interesting fact that these, more than any other operations upon the eye, excepting those for cataract, have given rise to so many modifications. Almost every year some new procedure is advocated. Surgical interference in these diseases does not imply any danger to the sense of sight itself, but this morbid condition must be removed, in order that the normal function of the eye may be entirely re-established as far as this is possible.

Bearing this in mind, the first point to be considered is **the preservation of the eyelashes,** and secondly, **the avoidance of a rough cicatricial eyelid which would constantly irritate the eye.** Furthermore in the choice of any particular operation, it is wise to consider what influence the method in question would have, as regards the prevention of a relapse, and this is certainly not easy to decide.

The most important point of all to be considered is the degree of intensity of the original morbid process. If the latter has almost died away, it is not likely that the trichiasis and distichiasis will increase. In such cases there is always more or less atrophy of the connective tissue. On the other hand, where trachoma and intense swelling of the conjunctiva and lid exist, it is quite probable that the secondary trouble will gradually get worse after the operation, no matter what method has been used. We are also seldom able to choose the most favorable time for operation. The trachomatous inflammation, pannus, and infiltration of the cornea are made worse by the irritation of the faulty eyelashes, and we are forced to interfere if we wish to effect a cure. Patients often do not care to wait or cannot come again. Furthermore we must confess that in no case of distichiasis can we predict with certainty that there will be no relapse. Observation of a number of cases extending over a period of years is quite convincing upon this point, and patients who have been operated upon, when seen at a subsequent period, not infrequently exhibit the same trouble more or less pronounced according to the circumstances. This is also the case with those treated by other surgeons. This tendency to relapse is more noticeable when Crampton's or Anagnostaki's method of operation is used. On the other hand, the continual irritation of the lid may cause new abnormal lashes to sprout in such a position that they come in contact with the cornea. This should not be considered as a real relapse, but is to be regarded rather as a continuation of the original trouble. Moreover in such cases the little hairs are usually isolated and can be easily removed, so that the result obtained by the operation is practically the same. Lately however I saw a patient afflicted with complete distichiasis, notwithstanding the fact that she had been operated upon for the same trouble ten years before. The method used in this case was probably that of Anagnostaki with Snellen's modification, which was in vogue at that time. I operated upon both of this patient's eyes, using the method to be described below.

Before passing to the consideration of my method of operation, I fear that my colleagues, having reached this point, will not be inclined to continue their perusal of this paper; this is not singular, for most of us are tired of hearing of new operations of this kind. Almost every one has his own procedure, and pretends to be satisfied with it. Even in the clinical lecture it is no longer possible to discuss and compare all the operations which have been proposed for entropium, distichiasis and trichiasis. For this reason I think it is important to select a method which adapts itself to the wants of the out-door patient, for we often meet cases which cannot be subjected to clinical treatment. Especially in regions where trachomatous affections are endemic, patients are found who will not undergo an operation (or postpone it indefinitely) unless they are treated upon the spot. What a disadvantage this is to such people! The local physician hesitates to perform the operation himself, because of the lack of an easy method and because there is no skilled assistant, and it is not possible to oversee the after treatment, aided by trained nurses. Let me hope therefore that I may be permitted to briefly discuss the principal methods having a bearing upon this subject. I wish to call attention to the fact that most of them require careful after-treatment, and also to add a few remarks concerning the indications for the different procedures. The usual operations are described in the text-books, but the subsequent modifications are probably not to be found there. Most of the latter are mentioned in Prof. Stellwag von Carion's[1] paper, which I cite here, as I do not wish to pass by the operation and observations contained therein.

It is well known that there are two principal methods for the cure of entropium, distichiasis and trichiasis, without distinguishing sharply these diseases, one from the other. The idea of one of these methods is to turn out the lid away from the eye, while in the other the edge of the lid is so drawn up that it no longer comes in contact with the eyeball.

Referring to the first of these two, as regards the upper lid, it consists in cutting out a horizontal strip and then bringing the edges of the wound together. This was not entirely efficacious, so Crampton modified the operation by adding two lateral perpendicular incisions, 6 to 8 *mm* long, passing through the entire thickness of the lid, one on each side near the angle of the eye, and also a horizontal incision on the conjunctival aspect, passing through the cartilage about 2 or 3 *mm* from the edge of the lid. The faults of this procedure have been discussed at length, and I will only add that it requires long after-treatment in order to retain the movable middle piece thus made, in such a position as to turn the lid away from the eye, after the wound has healed by granulation. Streatfeild's method, modified by Soelberg-Wells and Snellen, seems more rational. A prismatic strip, running down close to the conjunctiva, is cut out of the entire length of the cartilage, and the wound gaping widely above is closed by sutures. The cartilage is reached by incision through the skin and separation of the muscle fibres. This is a very pretty operation, and turns the lid well away from the eye, especially in entropium combined with curvature of the cartilage, but in real distichiasis it is not indicated, and is moreover full of details and requires skilled assistance.

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[1] *Allg. Wiener med. Zeitung*, 1885, No. 49.

Transplantation upwards of the edge of the lid has also given rise to numerous variations: the simpler method consisted in splitting the lid into anterior and posterior portions, and then sliding up the former (carrying the eyelashes) several millimetres, and fixing it there by means of sutures. This operation was done by Arlt and v. Graefe, each, however, having his own modification. The objection to this is that the naked border of the eyelid, several millimetres wide, stripped of skin and muscle, heals very slowly by granulation, and rapid ambulatory treatment is out of the question. Of course there have been numerous attempts to cover up this bare place, and secure primary intention. With this in view, it was proposed to either utilize the skin in the immediate neighborhood, or else borrow it from some other part of the body. The first method has found few partisans. Dr. Spencer Watson likewise split the lid in two portions, and then made two horizontal incisions in the anterior leaf, one 2 *mm* from the border of the lid, and the other 2 *mm* above this, thus dividing the anterior part into two strips; these strips were then detached at one end on each side, and their positions changed, so that the lower one with the lashes was twisted up above, while the upper was brought down near the edge of the lid.

After Riverdin had demonstrated that freshly detached pieces of skin could be easily transferred from one part of the body to another part, from which the skin had been removed, on to a granulating surface, this experience was soon applied to the operation under discussion. I had already proposed years ago to cover the raw surface left by the operation of Jaesche-Arlt with a thin slice of epidermis, removed by means of a razor from the inner side of the upper arm. This gives a good result, and the delicate scale of skin soon becomes adherent to the underlying structures. Careful after-treatment is indispensable, however, and the patient must be kept perfectly quiet. Story cuts out his flap from the mucous membrane of the mouth, and transplants it then to the raw place in question, and in this way avoids a new growth of eyelashes, the only disadvantage, however, being the unavoidable thickness of the piece of mucous membrane. Professor Stellwag von Carion[1] recommends a very simple and convenient method. He first splits the lid, as above described, into anterior and posterior layers, and then cuts off a strip from the former, about 4 to 5 *mm* in width, containing the eyelashes, and running off to a point towards the angles of the eye; this he then re-applies to the raw surface, with the hairs pointing upwards. No sutures are used, but the whole is covered over with a piece of tin-foil, smeared with some fatty substance, and a binocular bandage. It remains to be seen whether the eyelashes are preserved, and remain in a good position. I also specially emphasize the fact that Stellwag v. Carion himself recommends "complete bodily rest in bed for two or three days" after the operation. The same is true of all the other methods mentioned here, not excepting that of Professor Jacobson. The latter covers the exposed spot with a suitable flap, twisted around from the temporal region, and still connected with the remaining skin by a pedicle at the outer angle of the eye.

Bearing the above disadvantages in mind, I trust it may not seem superfluous to suggest an operation which is specially adapted to the wants of out-door cases, and which may be performed without skilled aid. The method is not entirely new, having been originally proposed by my brother, but it differs from his procedure in its most essential points. The most important difference is the employment of an instrument, the fundamental principles of which were suggested by Desmarres' lid-clamp.

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[1] "Ein neues Verfahren gegen einwärts gekehrte Wimpern."—Von Professor Stellwag von Carion, *Allgem. Wiener med. Ztg*, 1885.

This instrument consists of a plate, to be passed under the upper lid, and a ring-shaped piece, which rests upon the surface of the lid; these two parts are connected by an arrangement like a forceps (Fig. 1). The lower arm of the instrument ends in a round curved plate, 1 *mm* thick and measuring 24 *mm* from side to side at its broadest point, and 18 *mm* from before backwards (Fig. 2). The apex of the curvature is about 4 *mm* from the plane of the arm, to which it is attached. Just above the middle of this plate there is a slightly curved transverse notch, 1.5 *mm* wide, its anterior border 18 *mm* long, and its posterior border 21 *mm* long, the two being therefore joined together at an acute angle. The curvature of the notch has a radius of 3.9 *cm*. The upper arm ends in an oval ring, curved in the same manner as the lower plate, so that the two surfaces may be closely adapted to each other. The upper side of this ring is 1.5 *mm* wide; its lower side, where it comes in contact with

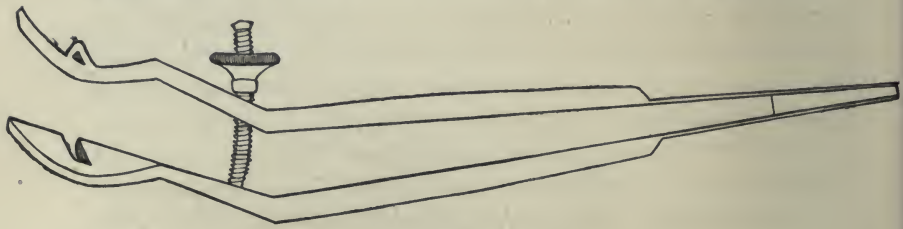
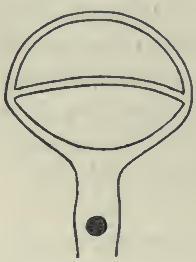


FIG. 1.



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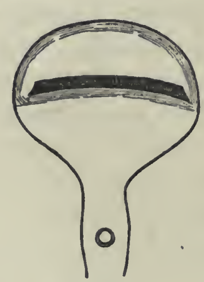

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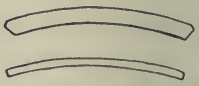

FIG. 4.

the lower plate, 0.5 *mm* wide. It is 2 *mm* thick (Fig. 3). Fig. 4 shows the end pieces, seen from in front.

The ring is bridged over from side to side by a bar which crosses it, of the same dimensions as the rest of the ring. The radius of curvature of this bar is the same as that of the transverse slit in the lower plate, and is so arranged that it corresponds exactly to the lower border of this slit when the instrument is closed.

The arms of the forceps are 12 *cm* long, and are bent upwards, at an angle of 35°, at their anterior fourth. Near this point a screw is fastened to the lower arm, which passes up through the upper arm, by means of which the oval ring may be brought in close contact with the lower plate. The instrument is nickel-plated and well polished, except where the ring comes in contact with the lower plate when the instrument is closed; these points are left rough.

In performing the operation with this instrument, either chloroform or cocaine may be used, the latter producing sufficient, but not complete, anæsthesia. I first disinfect the eye, and then inject about a quarter of a syringe full of an eight- to ten-per-cent cocaine solution, at the inner and outer angles of the lid. This causes a small swelling, which disappears when rubbed, and gives no noticeable trouble afterwards. Injection into the middle of the lid, on the contrary, causes a swelling which hinders the further progress of the operation. Several minims of cocaine are dropped into the eye at the same time. The operation may be begun a few minutes afterwards, and its stages are as follows:

1. The lid-clamp having been immersed in 1:8000 corrosive sublimate, is introduced by passing the lower plate under the upper lid in such a manner as to bring the cross-bar of the ring about 1.25 to 1.5 *mm* above the eyelashes. The projecting border of the lid should be grasped equally at all points so as to obtain the same width. If this is not the case, the lid should be pulled down or pressed forward when the instrument is screwed down, until the object in question is attained. The two end-pieces should be so firmly screwed down that there can be no slipping of the instru-

ment. The ring, where it comes in contact with the lid, is only 0.5 *mm* wide and presses it firmly against the roughened surface of the lower plate.

2. The operator draws the lid-clamp towards him, so that the end-pieces are horizontal and the tense lid is drawn away from the eyeball. A two-edged scalpel is then passed in perpendicularly through the middle of the lid, close to the upper border of the cross-bar of the ring, and allowed to make an incision 1.5 to 2 *mm* long, upon the conjunctival side; thereupon the wound is lengthened externally and internally by a probe-pointed bistoury, as far as the corner of the slit in the lower plate.[1] In this way, contact with the eye by a cutting instrument is avoided with certainty. The lower border of the cross-bar of the ring must be closely followed however.

3. The skin bounded by the upper segment of the ring is now removed, the incision commencing at the inner end of the horizontal cut and following the inner border of the anterior part of the ring until the outer end of the wound is reached. The skin within these boundary lines is grasped with the forceps and dissected away from the underlying tissue. The lid-clamp may now be passed to an assistant or simply allowed to hang down over the patient's face, thus keeping up the tension of the lid, and in no wise interfering with the execution of this part of the operation. If sponging be necessary, this also may be done by an unskilled attendant; as long as the instrument is firmly screwed down, however, hardly a drop of blood will be lost. After removal of the lid-clamp the horizontal incision through the lid, 21 *mm* long, will usually seem to be too short to raise the lateral parts of the ciliary border away from the eye. This is easily obviated by lengthening the incision laterally and medially about 1.5 to 2 *mm*, using a pair of blunt-pointed scissors and taking care to avoid the tear-duct. This leaves a small tongue of skin at the external and internal angles of the wound, which should be taken off with one snip of the scissors, so as to obtain a clean-cut,

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[1] I found Desmarres' knife (couteau mousse), invented by him for cutting the bridge of the cornea (which he allowed to remain until after the capsule was opened in cataract operations), very good for this purpose.

curved incision, passing from one angle of the eye to the other.

4. After the hemorrhage has stopped, and all coagula have been carefully removed, the detached eyelid should be raised, and coaptation with the skin of the curved incision secured by means of three sutures. The action of this transplantation of the lid has been misunderstood by many who have supposed the principal result was obtained by the shortening of the lid. This is not the case. When the detached lid is pushed up, *it makes a quarter of a revolution upon its long axis* and its upper or cut border is pulled backward and comes in contact with the raw surface of the lid, while the former anterior (skin) surface now looks upward, and the lower free border with the abnormal eyelashes is tilted forwards.

To obtain the most perfect result possible, the position of the transplanted border of the lid is of the utmost importance. For this reason, it is wise to note before beginning the operation, whether the epidermis of the lid is present in loose folds, or whether it is tightly stretched, or perhaps shortened by cicatricial tissue. If it is loose, the superficial skin of the lid may be entirely removed during the third stage of the operation, the flap then measuring about 7 to 7.5 *mm* at its highest point; should the skin, however, be tense, it is better to make the curve of the incision somewhat less marked, so that its highest point lies about 1.5 to 2.0 *mm* below the upper part of the ringed end of the lid-clamp. The bridge formed by the remaining portion of the lid should then be placed in such a position that its newly formed border exactly corresponds with the edge of the divided cartilage, and should be fixed there by means of sutures and an appropriate bandage.

Fine, moderately curved needles and antiseptic silk or catgut may be used for the sutures. The needle is first passed through the middle of the lower strip or lid-bridge, just above the lashes, and then two lateral stitches are taken. A piece of adhesive plaster 3 *cm* square is now pasted to the forehead just above the eyebrows, the sutures are tied in a single knot and thrown up separately upon the forehead, where they are held in position upon the plaster by an assistant. The operator then convinces himself that each suture is so disposed as to hold the lid-bridge in its proper position, taking care that the assistant does not allow any of them to slip. If in proper position, they are fixed by a second piece of plaster, covering the first piece, and then turned back over this and again roofed over by a third piece of plaster, in order to make assurance doubly sure.

If the incisions have been properly made, the border of the lid with the cilia is now in its proper position, the true lashes pointing somewhat upwards, while the abnormal hairs are directed forwards. The transplanted lid-border, about 2 to 2.5 *mm* wide, or perhaps slightly wider, adheres readily to the underlying tissue, and the lashes are permanently held in position. A protective bandage is necessary, however, in order to make sure of union by first intention. I usually use a piece of protective material (carefully cleaned with corrosive sublimate), which is so disposed as to press the lashes upwards, and put over this a perfectly clean piece of gauze or linen, covered in by a large wad of salicylated cotton. The whole is held in place by a monocular gauze bandage, wrung out in four-per-cent. carbolic acid. Stellwag von Carion covers the transplanted flap with a piece of tinfoil smeared with some greasy substance, the idea being to avoid disturbance of the transplanted part when the bandage is removed.

After-treatment: If the operation has taken place in the morning, the bandage may be changed that night; otherwise it may be left until the next morning. The dressing is usually found soaked through with a bloody discharge. After this it may be changed every twenty-four hours, or if this is not practicable, every forty-eight hours. The parts should be thoroughly cleansed each time, with bichloride or a borated solution of salicylic acid. The sutures must be taken out the second day, or the third day at the latest, but a light bandage with zinc or lead salve is still kept on until perfect union has taken place. If the patient remains under the observation of the physician instead of going home directly after the operation, cold applications may be made for a couple of hours, or a temporary bandage may be applied and an ice-bag laid upon this. This method of operation may seem full of complex details to the reader, but it is simple in its execution and gives a sure result. The edge of the lid is somewhat thick at first, but this disappears in the course of time. In some cases, where too much skin has been removed, the transplanted lower border or lid-bridge is raised too high, or after the supporting sutures are removed, the projecting edge of the exposed cartilage may not be entirely covered, and longer after-treatment is necessary; the granulating surface should be frequently touched with a stick of nitrate of silver.

As regards the lower lid in entropium and distichiasis, I take this opportunity to recall attention to a method proposed by me seven years ago.[1] I have modified this operation somewhat, and have always found it simple and very efficacious. Hotz's method also accomplishes the end in view, but it is not so simple.

I will illustrate my operation with the accompanying plates. Fig. 5 shows an eye with the lashes of the lower lid turned in, so that they rest against the eyeball. A curved needle, armed with a long catgut suture, is inserted at point 1 (Fig. 5) exactly under the middle of the lid and about 1.5 *cm* below it. It is then shoved through the connective and muscular tissue under the lid, following the course indicated by the figures running from 2 to 8, the needle passing out of the flesh and in again as each point of the series is reached, until it finally emerges at 8, close to 1, the original point of entry. Between these eight points there lies an oval piece of skin, which is cut out with the aid of Himly's fenestrated forceps.

Fig. 6 represents the eye after the operation has been completed. The end of the catgut (*b*) is passed from **above downwards** through the loop formed by the emergent suture between 4 and 5, while the other end (*a*) is carried through the same place from **below upwards.** By gradually pulling upon the two ends, a threefold result is obtained: **a considerable area of connective and muscular tissue lying under the lid is drawn together** like the mouth of a pouch; secondly, **the superficial incision is closed**; and thirdly, **the edge of the lid,** more particularly its middle part, **is turned outwards.** A permanent result is assured, owing to the firm cicatricial tissue, which forms in consequence of the squeezing together of the tissue by the catgut suture. The skin is excised in order to avoid unnecessary puckering when the suture is tightened.

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[1] *Klinische Monatsblätter f. Augenheilkunde*, December Heft, 1882.

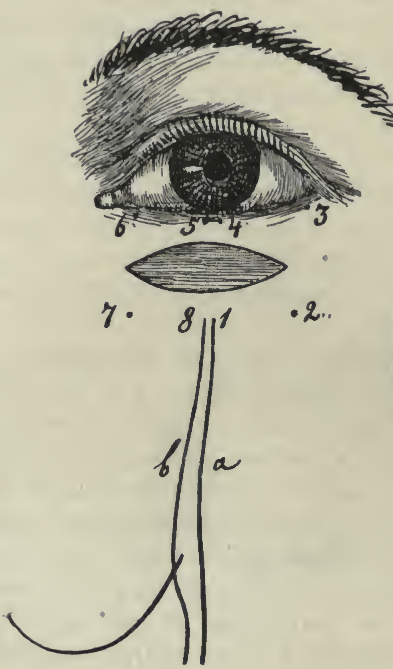

FIG. 5.

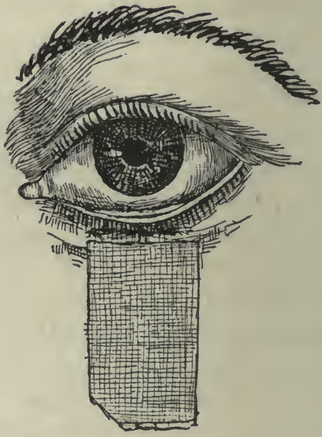

FIG. 6.

After the wound has been drawn together, the two ends of the suture should be tied in a single knot with a piece of silk thread interposed, so that it may be easily loosened when the dressing is taken off, which otherwise would be difficult, owing to the excessive swelling of the parts. A piece of adhesive plaster 3 *cm* long is now fixed to the cheek just below the wound; the four ends of the suture (two silk and two catgut) rest upon this, and are held in place by a second strip of plaster. The suture may be removed after four or five days, when the wound is healed, while the swelling gradually disappears. Out-door patients should be provided with a protective bandage.

A FEW REMARKS CONCERNING THE TREATMENT OF STENOSIS OF THE LACHRYMAL DUCT.

By Dr. E. JAESCHE, of Dorpat.

(With four illustrations in the text.)

Translated by Dr. J. M. Mills, New York.

RECENTLY Benson made the statement that in order to keep the passage-way of the lachrymal duct patent, after the stenosis had been relieved, he used a lead style, which the patient inserted at night. I quite agree with him, as I have employed the same method for many years. But there are always dissenting voices which recommend an obliteration in severe inflammation of the lachrymal duct. Therefore Eversbusch proposes, in chronic catarrhal inflammation of the lachrymal sac, first to destroy the lachrymal duct with the galvano-cautery, then to extirpate the sac with a sharp spoon, and finally to remove the lachrymal gland. I maintain, on the contrary, that I have succeeded in making the passage of the lachrymal duct patent again in all cases of disease of the lachrymal passage, even when a fistula already existed, except in those cases where the surrounding soft parts had been excessively changed by disease, or the duct obstructed by bony proliferations. If the lachrymal duct is kept open, success is more likely to be obtained in curtailing the inflammation and the unhealthy suppurative secretion, and in curing the fistula.

It would hardly appear opportune to enter into a discussion of the manifold procedures used in stenosis of the lachrymal duct. I will therefore only cite a case that offered an opportunity for long-continued observation, and that may serve as an example.

Mrs. N., aged twenty-seven, came to me fifteen months ago, with the complaint that her left eye caused her a great deal of annoyance, prevented her from attending to her duties as a teacher, as it teared constantly and was often inflamed. Ten years ago she suffered with a severe inflammation of the lachrymal sac, with perforation of the abscess externally. She was treated at that time, and an attempt was made to restore the passage of the lachrymal duct. As, however, after several trials, they were still unsuccessful, further treatment was abandoned, nor was it attempted again. Since then there has been no repetition of the inflammation of the lachrymal sac, the condition of which has remained the same.

Upon examination, a thinning of the skin over the lachrymal sac was noticed, some sinking in and white decoloration from the cicatricial tissue. The anastomosis of the canaliculus lachrymalis with the sac had become adherent. The patient entreated me, if it was at all possible, to reopen the lachrymal duct, and referred to another of my patients upon whom an operation performed for that purpose was successful. Naturally we would hesitate to operate in such a case, as the existing condition is considered by many to be the most fortunate issue of a suppurative dacryocystitis, and they even endeavor to produce such a result by obliteration of the lachrymal canal. I informed the patient of the uncertainty of the result, assured her, however, if she positively desired it, that I was willing to make the attempt to reopen the lachrymal sac.

Before the operation I instilled a few drops of a strong solution of cocaine into the inner corner of the eye of the diseased side, then used my silver conical-grooved sound with the accompanying small knife.

If the entrance into the lachrymal sac is unobstructed, I prefer to use Stilling's knife for division of the adhesions in the lachrymal canal, after a preliminary division of the canaliculus; but when the inner end of the lachrymal duct is already closed, I prefer to use the instruments, which I have previously proposed.[1] The sound (Fig. 2) is provided above with a plate, in order that it may be more conveniently held. It tapers towards a point, and on one side has a groove, which extends almost to the point (Fig. 1). The sound is slightly curved for each side of the head, in such a manner that when erect the groove is turned outwards. This position is obtained when the sound is inserted horizontally into the (slit) lachrymal duct and then elevated so that it stands perpendicularly above the lachrymal sac. Then the sound is pressed down until we are sure we have penetrated through the sac, into the nasal passage, or at least into the sac. Then we hold the small knife, which, for the sake of convenience, is provided with a double curvature in the plane of the edge and of the flat surface, in an appropriate manner for each side.

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[1] *Graefe's Arch. f. Ophth.*, Bd. x., S. 170.

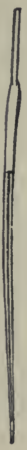
FIG. 1.

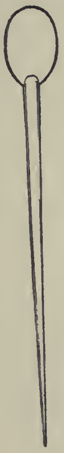
FIG. 2.

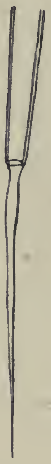
FIG. 3.

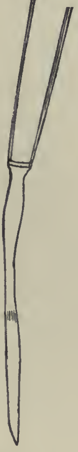
FIG. 4.

(Fig. 4 shows the knife as seen from one side, Fig. 3 as viewed in the plane of the edge, both in the curvature intended for the left lachrymal duct). This knife is inserted into the groove of the sound so that its sloping end can slide down while the extreme end remains covered. When the knife has reached the lower end of the lachrymal sac, the sound is removed, and the knife, carefully guided by the touch, is pressed forward into the nasal passage. Obstructing adhesions and structures will be divided by elevating and depressing the knife, and at the same time turning it sidewise. Upon withdrawing it, the part where the lachrymal canal anastomoses with the sac is somewhat more extensively divided. There is usually only a slight hemorrhage. If the reopening of the lachrymal duct has been successful, then Weber's stricture-knife is inserted in order to ascertain how much of the lumen of the duct has been restored, and if it appears insufficient I widen it by strong pressure with the sound.

In the present case, in performing the procedure described, the lachrymal duct was found to be pervious and could be satisfactorily widened. After the cessation of hemorrhage, a piece of the strongest Italian violin catgut (B), hastily washed in a sublimate solution, was introduced into the duct. Such a piece, 3 to 3.5 *cm* long, is carefully rounded off with a file at the lower end, and at the upper end it is slightly notched, so that it can be furnished here with a thread. When the sound has penetrated through the lachrymal duct into the nasal passage, the ends of the thread are carried up and fastened to the forehead, between the eyebrows, with a piece of English plaster. With some patients one may use a somewhat thicker string, the thinnest cello string (A).

The catgut swells and prevents a re-union of the divided parts and maintains a uniform dilatation of the duct. It often causes some pain, varying in intensity, in the first few days, if there is much swelling, so that some patients remove the catgut after three, four, or six hours, whilst others let it remain until the next morning. To remove the string it is only necessary to pull on the loosened thread. In order to quiet the pain due to the tension, after the catgut has been introduced, and also later, when it has been removed, cold applications may be used. In a few days the irritability of the wound is allayed so that the string can certainly be left in overnight. It is then advisable, immediately after the removal of the catgut, to commence syringing with a disinfecting or a slightly astringent solution. If the patient removes the string in the morning, after thoroughly cleansing the eye, he can instil a drop of the afore-mentioned solution.

The catgut is used daily, in this manner, for several weeks, and not exchanged for the lead style until the irritation in the lachrymal sac has entirely ceased. If the treatment we have described is undertaken after a severe inflammation of the lachrymal sac with, perchance, perforation of the abscess externally, then it may last several weeks, and perhaps it will be necessary for a time to use warm applications.

Miss N. could retain the catgut for several hours in the first few days after the operation, later on, until late in the evening, and then until the next morning. Right in the beginning the anterior wall of the lachrymal sac, formerly sunken in, became swollen at the place where there had been a fistula at the time of the first inflammation. This swelling, at one time larger, at another smaller, was not entirely reduced until several months had elapsed; it was frequently painted with tincture of iodine. As long as this lasted of course I forbore syringing the lachrymal duct, only instilled a solution of zinc sulph. and boracic acid. The patient removed the catgut early in the morning and a new one was introduced in the evening. Only after three months was it considered advisable to pass over to the use of the lead style. These I prepare myself, for each case, in the proper length and thickness, by working a piece of lead wire. The diameter of the style varies from 1.5 to 2 *mm;* the upper end is bent in so as not to annoy the eye, while it gradually tapers at the lower end. In the case in question quite a thin style was used. I had to insert it for several weeks, but gradually the patient learned how to introduce it, which she did before retiring.

Miss N. was very well satisfied with the success of the treatment; her eye gradually succumbed less and less to the recurring irritation, although the tear-conduction was not entirely restored to its normal condition. Once, after about a year had elapsed, the eye, with the lachrymal duct and Schneiderian membrane, owing to an injury, again became inflamed. Once more there was epiphora and a muco-purulent secretion in the lachrymal duct. With the appropriate treatment and an occasional use of the catgut, instead of the lead style, this inflammation soon disappeared.

The patient was of the opinion that she felt better when she allowed the style to remain in overnight instead of during the day. With some other patients it was possible finally to do without the style altogether, as the lachrymal duct remained open without it. I would only add that when a satisfactory result can be obtained in stenosis of the lachrymal duct with Bowman's sounds, naturally these are to be preferred to any more heroic procedure.

THE VISUAL DISTURBANCES IN TABES DORSALIS, AND AN ATTEMPT AT A COMPREHENSIVE EXPLANATION OF THE SYMPTOM-COMPLEX OF TABES.

By Dr. E. BERGER, Paris,
FORMERLY DOCENT AT THE UNIVERSITY OF GRAZ.

Translated by Dr. Ward A. Holden, New York.

I PUBLISH the following investigations with the consciousness of the great responsibility assumed in attempting to solve problems of such importance. I believe it, however, the duty of every investigator to present his results free from bias. The material that I have studied is, moreover, not so small that the results can be considered accidental.

While later investigations in one or another detail may give results which will modify my views, I believe that these investigations will at least offer a stimulus to further study of the nature of the tabic process.

To all those who have assisted me in my work, especially Charcot, Déjerine, Ed. Meyer, and Parinaud, of Paris, who placed their material at my disposal, and Brown-Séquard, in whose laboratory I carried out the experimental studies, I wish here to express my thanks.

The Symptom-Complex of Tabes.

As is known, the symptoms of tabes, besides those caused by disease of the spinal cord, consist of disturbances of function of special cerebral nerves, sometimes of the cerebrum, and probably as well of the cerebellum.

The symptoms of disease of the spinal cord are : lancinating pains, paræsthesia, hyperæsthesia, and, later, dulled sensation of the skin, lessened sensibility to pain, and hyperalgesia. The muscle function is disturbed in the beginning in its co-ordination, later in its motor strength. The tendon reflexes are wanting. The bladder and the genital organs suffer in their function. On the part of the digestive tract, gastric crises and constipation are frequent symptoms. On the part of the vascular system, an increase in the frequency of the pulse is often observed. Probably the hyperidrosis of particular regions of the skin is associated with an altered activity of the vessels. In the later stages of tabes, trophic changes take place in the skin (herpes, lichen, pemphigus, decubitus), and in the muscles, bones, and joints.

Of the general disturbances of the brain should be mentioned : headache, disturbances of speech, failure of memory, and disturbances of intellect ; yet these are unusual. Most frequently particular cerebral nerves are affected, the optic nerve, the nerves of the ocular muscles, and single branches of the fifth. It is seldom that the hypoglossal, the glosso-pharyngeal, and the olfactory nerves are also affected.

Besides these are reported the occurrence of diabetes mellitus and insipidus, of bronchial asthma, of nephralgic crises, and of rectal and urethal colic.

Certain disturbances of the sense of balance (Romberg's symptom) would indicate that the cerebellum may also suffer in its functions.

The question how this symptom-complex is to be explained, what primary process can be followed by affections of parts of so different a nature, has hitherto been answered in a variety of ways. If one consider all the symptoms of tabes as co-ordinated manifestations of the disease (Gowers[1]) or if one call tabes a "combined system-disease" (Strümpell[2]), these are, properly speaking, no explanation, but a mere statement of existing facts.

The Pathological Changes in Tabes.

Pathological changes have been observed in tabes in the medulla oblongata, in the peripheral cerebral and spinal nerves, in the spinal ganglia, and in the sympathetic nerve.

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[1] Gowers, Brit. Ophth. Soc., June 7 and 8, 1883.

[2] Strümpell, Die path. Anatomie der Tabes dorsalis, *Arch. f. Psychiatrie*, vol. xii., 1882.

In the cord, particular fibre regions are typically diseased in tabes, while fibres of other physiological function, as in the case of poisoning from lead or ergot (in the beginning), are not affected.

The histological changes are, in the beginning of the disease, a thickening of the interstitial tissue, an increase in nuclei, an enlargement perhaps of the nerve cells themselves, later the destruction of the nerve fibres, and the appearance of granular cells and corpora amylacea.

The walls of the vessels appear thickened and rich in nuclei. We shall return to this subject later.

Erb supposed these changes to be the consequences of a chronic inflammatory process, which shows itself in a degeneration of the nervous elements, and in an irritation and an increase of the interstitial connective tissue; according to Charcot, it is a parenchymatous inflammation of the nerve substance, and the changes in the interstitial connective tissue are of a secondary nature.

We know from the investigations of Charcot and Pierret that the columns of Goll are the parts of the cord first affected. The process, which in its early stages affects the "bandelettes externes" of the posterior columns (Charcot and Bouchard), has its greatest extent in the upper lumbar and dorsal portions of the cord, and higher and lower down the affected region is smaller in the cross-section. The pia mater corresponding to the diseased parts is thickened and opaque.

The pathological process may extend upward into the restiform bodies and the medulla, or spread laterally into the posterior cornua, the lateral tracts, and the columns of Clarke. In the severest cases the disease may invade the whole cross-section of the cord.

Strümpell, in his investigation of cases of incipient tabes, found in the dorsal portion of the cord a degeneration of two narrow, lateral, symmetrical fields in the posterior columns, into which passed fibres of the posterior cornua. In addition, there was a degeneration of a narrow band on either side the posterior fissure.

In the lumbar region he found degeneration of the posterior root fibres of moderate extent. In the cervical region the columns of Goll were diseased, first in the posterior, then in the anterior portion.

An accurate statement of our present knowledge of the extent of the affection of the cord may be found in the paper by Martius (*Berliner klinische Wochenschrift*, 1888).

The investigations of Pierret first threw light on the nature of the connection between the changes in the cord and those in the medulla, of which latter we shall speak hereafter.

Pierret brings the symptoms of tabes into connection with an intermediary fibre-system of motor and sensory zones.

This system forms, where the auditory and facial appear, a nerve, the nervus intermedius Wrisbergii, which springs directly from this ascending, mixed bundle (solitary fascicle, Stilling), that receives motor and sensory fibres from the glosso-pharyngeal nerve and the vagus. Pierret found that at the level of the decussation of the pyramids, this bundle bends in an arc with its convexity outward, reaches the spinal-accessory nerve, and forms in the cord an intermediary zone of sensory and motor fibres; it connects with the ascending fibres which occupy the cervix cornu posterioris and the deep part of the lateral tracts; here lie also the vasomotor fibres which spring from the intermedio-lateral tract, which latter is very often affected in tabes, when a series of vasomotor disturbances result.

Hadden described (1882) in tabes a new degeneration, which from the cervical portion of the cord ascends into the medulla, and which lies in the anterior part of the anterior columns directly outward from the gray substance. This was followed to the olivary bodies.

Gowers found (1886) in tabes a degeneration of an antero-lateral tract, which, at the height of the third cervical vertebra occupies the same position as lower down, and also extends as a very thin band between the cerebellum and the pyramidal tract near the surface of the cord. In the lumbar region the tract lies almost entirely in the lateral, and anterior to the pyramidal tract.

In tabes changes have rarely been found in the medulla, perhaps because the necessary attention was not paid to the examination of this part. Erb says of the oculomotor, abducens, and trochlear nerves: "The nuclei of these nerves in the floor of the fourth ventricle also seem to be affected in many cases." A chronic ependymitis with granular hyperplasia seems to occur very often at this point in tabes. Among others, Magnan has described this condition very accurately in two cases of tabes in alcoholic subjects.

As to the nuclei in the floor of the fourth ventricle, we find statements that these have been found intact. Oppenheim remarked respecting a case with laryngeal crises: "I can still say with certainty that those parts in the medulla oblongata which are connected with the vagus, viz., the vagus nucleus, the longitudinal fibres of the pons, and the vagus roots, were found intact."

Opposed to these are direct observations of affection of the nuclei in the floor of the fourth ventricle.

Buzzard[1] in 1884, in sections from a case of tabes, found in the medulla, disease of the funiculi cuneati and graciles, the cerebellar tracts of the restiform bodies, the outer portion of the vagus nucleus and the solitary fascicle.

Landouzy and Déjerine[2] in sections from a tabic woman, æt. fifty-four, who suffered from laryngeal crises and was blind, found the roots of the vagus and the spinal accessory reddish-gray, and their nuclei showed a pigmentary atrophy of the cells, especially in the upper part. The hypoglossal nucleus, the roots of the descending branch of the fifth and of the facial nerves, were normal. The sclerosis of the posterior columns extended to the calamus scriptorius.

Kahler examined the cord of a patient æt. forty-seven, who suffered from paralysis of accommodation, paræsthesia of the legs, girdle sensations, left-sided ptosis, difficulty in swallowing and in micturition, paresis of the lower facial and of the right vocal cord. The pulse was increased in frequency. The sections showed an almost total degeneration of the posterior columns in the lumbar and dorsal regions, and a degeneration of the columns of Goll in the cervical portion.

In the floor of the fourth ventricle were found chronic meningitis and ependymitis. Underneath the ependyma a sclerotic process had extended into the nerve substance itself. The right posterior vagus nucleus was totally, the left partially, atrophic. The abducens and facial nuclei were also involved.

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[1] Buzzard, *Brain*, January, 1884, pp. 461–486.

[2] Landouzy and Déjerine, Société de Biologie, May 19, 1883.

Similar conditions are reported by Jean and by Otto.

In one case of Buzzard of multiple paralyses of the exterior ocular muscles which Bevan Lewis investigated, degeneration of the abducens nucleus and numerous hemorrhages in its neighborhood were found.

As for *disease of the optic nerve*, no direct connection with that of the medulla or cord has hitherto been shown. On the contrary, *the changes begin constantly in the optic nerve* (Leber[1]) and not in the tract, with which clinical facts agree (Förster[2]). According to Leber the disease appears first in the periphery of the optic nerve and affects the central fibres later.

It begins constantly in the portion of the nerve near the eye and extends centripetally into the tract, and in a few cases even to the corpora quadrigemina.

Charcot was able in one case to follow the degeneration to the corpus geniculatum externum.

Westphahl[3] in one case found the changes uniformly distributed over the cross-section of the nerve. E. von Jaeger found the intervaginal space of the optic nerve widened into a broad circular sinus.

Poncet[4] had an opportunity of studying the optic nerve of a tabic patient blind for ten years. The sheaths of the optic nerve and its connective tissue were considerably thickened. The nerve fibres were changed into an amorphous granular mass. The intracranial part of the optic nerve showed no change in its connective tissue, but was however, in a condition of fatty degeneration.

In the retina, Leber found the nerve fibre and the ganglion cell layers atrophic and the tissue supporting the vessels slightly thickened.

Changes were found also in the marginal fibres of the sympathetic nerve by Raymond and Arthaud,[5] pupils of Vulpian. They found increase of connective tissue to be the chief cause of the atrophy of the ganglion cells, which began with the formation of fat drops and the increase of pigment, and led to complete destruction of the cells.

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[1] Leber, *Graefe's Archiv*, vol. xiv., 2, 1868, p. 177.

[2] Förster, *Graefe and Saemisch's Handbook*, vol. vii.

[3] Westphahl, *Archiv f. Psychiatrie*, 1884, vol. xv., part 3.

[4] Poncet, Société de Biologie, Feb. 18, 1882.

[5] Raymond and Arthaud, Société de Biologie, July 23, 1882.

In the meeting of the Academy of Sciences at Paris, July 28, 1886, Charcot reported the investigations of Pierret, which demonstrated the frequency of peripheral neuritis in tabes. This neuritis is not constant, it may get well. Pierret believed that the existence of this neuritis in connection with a central lesion justified the acceptance in nerve pathology of a new sort of inflammation, which showed diseased foci with sound intervening tissue, in various parts of the affected system.

The occurrence of peripheral neuritis has been frequently observed since then.

In reality it would seem to be centrally the same process that Gowers (1879) found in a case of nuclear paralysis of the ocular muscles, which Hutchinson had observed clinically. He found in the nerve nuclei and in the nerve roots the same changes that are found in the anterior cornua of the cord and in the roots of the spinal nerves in progressive muscular atrophy. Hutchinson says that this indicates a sort of creeping neuritis, which attacks the nuclei of the motor nerves, spreads from one to another, and has a tendency to develop in distant parts.

Déjerine,[1] in a case of bilateral ptosis, found the nerve nucleus and trunk intact, but a peripheral degeneration of the branch of the nerve supplying the lid.

Pierret and Hayem demonstrated also that the peripheral portions of the fifth and of the auditory nerve might suffer in tabes.

Former Attempts at a Comprehensive Explanation of the Symptom-Complex of Tabes.

Duchenne explained tabes as a disease of the cerebellum, Neftel (New York) as a disease of the brain, since cerebral symptoms may occur in it.

Trousseau considered tabes a neurosis, Leyden conceived it to be an atrophic process in the nerves. Charcot, and after him Friedreich, thought it a chronic inflammation, with hyperplasia and fibrillar degeneration of the interstitial tissue. The optic was the cerebral nerve most frequently attacked, since its texture most resembles that of the central nervous system.

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[1] Déjerine, Société de Biologie, October 18, 1884.

C. Lange supposed a chronic meningitis of the nerve roots, from which the changes in the posterior columns develop, as a secondary degeneration.

Pierret called attention to the irritation of the region supplied by the fifth nerve, and explained the paralysis of the ocular muscles, etc., as a reflex paralysis.

Erb[1] demonstrated that the sclerosis of the posterior white columns might explain the lancinating pains, the paræsthesia, and the anæsthesia, while the affection of the lateral tracts might account for the motor disturbances. They would not explain the weakness of the bladder, the disturbances of the sexual function, and the trophic disturbances.

Takacz and Woodhead are of the opinion that the tabic process begins as a degeneration of the axis cylinders in the peripheral and central sensory tracts. The ataxy is the consequence of interrupted and delayed sensory conduction.

Wharton Jones[2] remarks that years after a spinal injury, amaurosis may develop. Paralysis of the sympathetic nerve may first lead to a hyperæmic condition, and later to atrophy of the optic nerve.

Albutt, Arndt, Waldmann, and N. Weiss[3] suppose that a chronic meningitis precedes, and that the disease of the cerebral nerves is caused by the spreading of the process to the base of the brain.

Förster[4] comes to the conclusion that the gray degeneration develops simultaneously or successively at various predisposed points in the central nervous system.

Rieger and Förster[5] seek the conection between the spinal disease and the optic atrophy in the sympathetic nerve.

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[1] Erb, *l. c.*, p. 201.

[2] Wharton Jones, *Brit. Med. Jour.*, July 24, 1869.

[3] N. Weiss, *Wiener Klinik*, 1880, No. 6.

[4] Förster, *Graefe and Saemisch's Handbook*, vol. vii., 1, p. 131, 1876.

[5] Rieger and Förster, *Graefe's Archiv*, vol. xxvii., 3.

The nerve atrophy depends directly on the part of the cord primarily affected, and arises through vasomotor disturbances.

Leber[1] believes that the cord has a direct influence on the nutrition of the optic nerve, and he founds this opinion on a case of Mooren's, in which atrophy of the optic nerve followed a blow in the loins.

Schmeichler[2] would explain the simultaneous existence of paralyses of ocular muscles and the cord disease by the vascular supply. "The arteria vertebralis supplies the posterior columns with branches, it supplies as well the root ganglia of the ocular muscles through the arteria cerebri superioris and the arteria cerebelli inferior and posterior."

Mauthner[3] supposes that the sclerotic process begins in the fourth ventricle and in the aquæduct of Sylvius, and thence continues to the thalamus; from this latter it propagates itself as a descending degeneration of the optic nerve.

The Impossibility of Explaining the Symptom-Complex of Tabes by the Theories Offered.

The former attempts to prove a direct anatomical connection between the affection of the optic nerve and the disease of the cord have been unsatisfactory. For the sclerosis of the lateral tracts the following course is supposed: restiform bodies, lower portion of the pons, peduncle, white substance, optic thalamus, internal capsule, and optic tract.

Michel[4] says: "If one could suppose a disease of the recessus, it must be admitted that the fibres running to the macula lie in the chiasm close under the floor of the recessus." This explanation is untenable, since, with few exceptions, central scotomata do not occur.

The supposition of the extension of the disease from the cord to the optic nerve through the medium of the sympathetic nerve cannot be admitted, since, according to Schulten,[5] this nerve has no direct influence on the fulness of the retinal vessels.

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[1] Leber, *Graefe and Saemisch's Handbook*, vol. vi., p. 872.

[2] Schmeichler, *Arch. f. Augenheilk.*, vol. xii., p. 472, 1883.

[3] Mauthner, "Die nuclearen Augenmuskellähmungen," p. 382.

[4] Michel, *l. c.*, p. 629.

[5] Van Schulten, cited in *Arch. f. Augenheilk.*, 1883.

The explanation of the symptom-complex by the assistance of a basilar meningitis is untenable: (1) because the latter has not been found at the autopsies: (2) because there is no reason why this particular one of the cerebral nerves should be attacked by the meningitic process, while the others are spared.

The conception of the functional disturbances of the cerebral nerves as reflex neuroses is not satisfactory, since nerve pathology has not recognized a reflex *atrophy.*

The explanation of Mauthner, that a chronic ependymitis extends forward on the wall of the thalamus is objectionable, since clinically there is seldom hemiopia, and the anatomical investigation has not shown a *descending* atrophy.

The explanation of Förster[1] that similar histological changes can develop simultaneously or successively at various predisposed points in the central nervous system, is, as he himself admits, unsatisfactory.

Attempt at a Comprehensive Explanation.

Under these circumstances, in my investigations on the visual disturbances in tabes, I considered it advisable to seek facts which would support a comprehensive explanation of the symptom-complex of tabes. I wish to state here in the beginning that the medulla oblongata seemed to me to be the connecting link between the spinal and the optic nerve affection.

Disease of the medulla undeniably exists in tabes. We know through Adamkiewicz[2] that the affected parts of the cord correspond to special vascular regions. It may be assumed that these vascular regions are affected (vasomotor disturbances are found) through disease of the nerve nuclei in the medulla.

Förster and Leber have also supposed the atrophy of the optic nerve to be a degeneration caused by vasomotor influences, all other attempts at an explanation seeming to them untenable on theoretic grounds.

I have proposed the question to myself in each case examined clinically, whether and in what portions the medulla might be diseased, and I have then compared the cases in which atrophy of the optic nerve existed with those in which it was wanting. From the position of the nuclei of the nerves most frequently affected in common with the optic, I have drawn conclusions as to the probable position of the vascular centre for the optic nerve, which I have later tested experimentally. It was necessary that these remarks precede in order that my methods of investigation be understood.

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[1] Förster, *Graefe and Saemisch's Handbook*, vol. vii., p. 133.

[2] Adamkiewicz, Die Blutgefässe des menschlichen Rückenmarks, *Wr. Sitzber.*, vol. lxxxiv., 1882.

My experiments on animals had shown me that disease of the medulla could explain the symptoms of tabes.

Canfield[1] observed a case which in vivo exhibited all the symptoms of tabes. At the autopsy were found a blood coagulum filling the fourth ventricle, traces of blood in the acquæduct of Sylvius, and another blood clot in the third ventricle.

Dr. Van Millingen, with whom I spoke in regard to my investigations in tabes, informed me that he had observed clinically a case similar to this in Constantinople.

The Influence of Age and Preceding Syphilis on the Frequency of Tabes and on the Development of Serious Ocular Complications in Tabic Cases.

My material for investigation consisted of 109 patients, 93 males and 16 females, a relation which allows no conclusion as to the relative frequency in the two sexes. 26 were in the stadium præatacticum, 50 in the stadium atacticum, and 33 in the stadium paralyticum.

As to the age at which the first symptoms of tabes appeared :

Age.	Total.	Luic.[2]	Non-luic.	With ocular muscular paralyses.	With atrophy of the optic nerve.
15–20 years	1	—	1	—	—
21–25 “	4	2	2	2	1
26–30 “	8	3	5	2	3
31–35 “	18	7	11	7	10
36–40 “	11	2	9	8	7
41–45 “	33	20	13	18	19
46–50 “	14	4	4	7	7
51–55 “	7	3	3	2	1
56–60 “	5	2	—	1	4
61–65 “	—	—	—	—	—
66–70 “	—	—	—	—	—
71–75 “	2	2	—	—	—

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[1] Canfield, *Lancet*, 1885, vol. ii., No. 3, p. 110.

[2] Cases suspected of lues are here included with the luic.

We may conclude from this tabulation of 106 cases—in three cases the date of the beginning of the disease could not be ascertained—that tabes is found between the ages of fifteen and twenty years, but not frequently; it becomes gradually more frequent and reaches the maximum of frequency betwen the ages of forty-one and forty-five; later in life it becomes less frequent (? in relation to the number of those alive at this age), but can appear even in old age, as two cases show, in which the disease developed after the seventieth year.

In general, my figures as to the frequency of tabes in different periods of life agree with those of other authors. Erb, however, found the maximum between the ages of thirty-one and forty years; Althaus between the ages of thirty and thirty-nine years; Topinard found an equal frequency between the ages of thirty-five and forty, forty and forty-five, and forty-five and fifty years.

At any rate, these tables show that tabes has its greatest frequency at the most vigorous period of life.

If we understand by the severe ocular complications of tabes, atrophy of the optic nerve and paralyses of exterior muscles (paralyses of accommodation are easily corrected by convex glasses), the following chart gives a graphic representation of their relations (Fig. 1).

In this figure the upper curve shows the frequency of tabes at the various periods of life; the middle curve shows the frequency of severe ocular complications at the various periods of life; the lower curve shows the frequency of atrophy of the optic nerve.

We find, from this chart, *that the cases of tabes occurring in the earliest and the latest periods of life are free from severe complications;* further, that the period of most frequent occurrence of tabes shows only a slightly increased percentage of severe complications. Severe complications arose in 66.5 % of the total number of patients, and in 69 % of the cases between the ages of forty-one and forty-five years.

As to the influence of a previous syphilis, I found:

Men— 44 syphilitic; 49 not syphilitic.
Women— 3 " 13 " "

Syphilis was therefore proven or assumed to exist in 43 % of the cases. In my material, so large a percentage of tabic patients having syphilis as Fournier (1876), and later Vulpian, Hutchinson, and Gowers found, did not exist. Quinquand found 100 %, Althaus[1] 86.5 %, Erb 90 % of tabic patients syphilitic. My figures nearest resemble those of Buzzard (45 %).

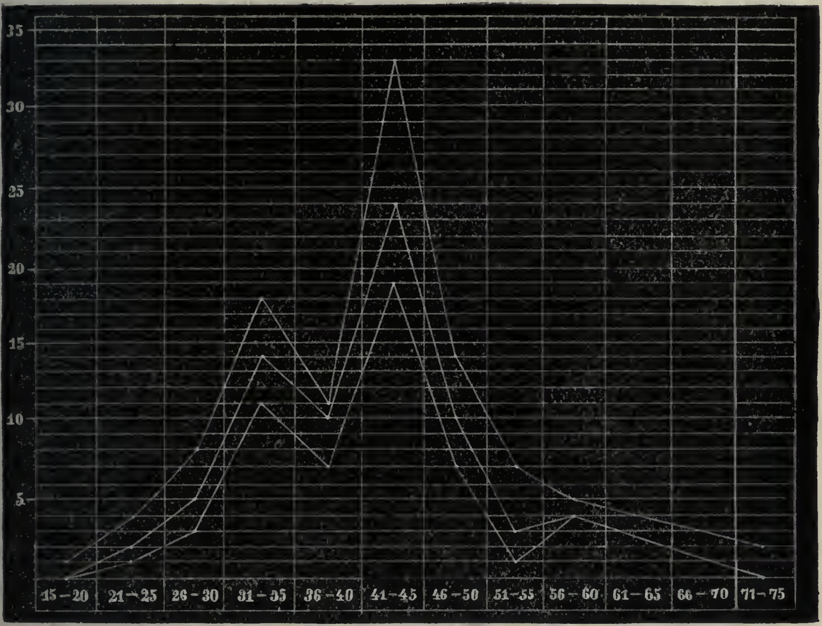

FIG. I.

Upper curve = Frequency of tabes.
Middle curve = Frequency of severe ocular symptoms.
Lower curve = Frequency of atrophy of the optic nerve.

We see by the preceding table that the earliest age at which tabes appears shows no primary syphilis; that the period from forty-one to forty-five years shows the absolute maximum of syphilitic cases and of non-syphilitic cases as well; and that the later periods of life show a relatively larger number of syphilitic cases.

The determination of the time which elapsed between the primary affection and the appearance of the tabes is of importance in regard to the question whether a relation exists between syphilis and tabes. This interval was determined in thirty-six cases.

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[1] Althaus, "Krankheiten des Rückenmarkes," French edition, 1885.

The tabes appeared after the primary affection :

In ½ year	in 1 patient	In 12 years	in 1 patient
" 1 "	" 3 patients	" 13 "	" 1 "
" 3 years	" 4 "	" 14 "	" 2 patients
" 4 "	" 4 "	" 18 "	" 1 patient
" 5 "	" 1 patient	" 21 "	" 3 patients
" 6 "	" 1 "	" 22 "	" 1 patient
" 8 "	" 6 patients	" 24 "	" 1 "
" 10 "	" 3 "	" 34½ "	" 1 "
" 11 "	" 1 patient	" 54 "	" 1 "

There were then affected with tabes:

Under	5	years after the primary affection of syphilis	......13 patients
Between	6–10	" " " " " " "	......10 "
"	11–15	" " " " " " "	...... 5 "
"	16–20	" " " " " " "	...... 1 patient
"	21–25	" " " " " " "	...... 5 patients
	later	" " " " " " "	...... 2 "

On an average the first symptoms of tabes appeared 11.5 years after the primary affection of syphilis.

As to the case in which tabes came on six months after the syphilitic affection, the interval is too short for the syphilis to have been the immediate cause of the tabes, as is accepted, among others, by Charcot and Krafft-Ebing.

As to the influence of a pre-existing syphilis on the initial symptoms of tabes, I have found that the tabes began in sixty-two cases with spinal symptoms (72.6 % non-syphilitic, 27.4 % syphilitic), and in forty-seven cases with cerebral symptoms (60 % syphilitic, 40 % not syphilitic).

This teaches us *that tabes in syphilitic cases begins for the most part with cerebral symptoms, in non-syphilitic cases for the most part with spinal symptoms.*

The cerebral symptoms were : headache with fever, drowsiness, tinnitus aurium, headache and nasal voice, transitory aphasia, fainting, gastric crises, and apoplectiform seizures.

The question now arises, whether a pre-existing syphilis causes a severity of the symptom-complex of tabes, *i.e.*, whether in these cases paralyses of the ocular muscles and atrophies of the optic nerve are more frequent.

Our statistics give the following information: the percentage of syphilitic patients affected with atrophy of the optic nerve was 43.4, and with paralyses of the ocular muscles 44, while the syphilitic cases formed 43 % of the total number of tabic cases.

It is evident then that syphilitic tabic patients are affected with severe complications almost as frequently as non-syphilitic. The question whether syphilis exerts an influence on the course or the period of development of the atrophy of the optic nerve and the paralyses of the ocular muscles will be considered later.

Symptomatology.

Width of the Palpebral Fissure.

In a number of tabic patients, where no paralysis of the oculomotor nerve existed, a *slight narrowing* of the palpebral fissure was noticed. This was described, I believe, first by Jacobson[1] as a symptom accompanying the myosis.

Altogether I found this slight drooping of the upper lid from paralysis of the sympathetic nerve in 42 cases, in 17 cases it was unilateral (3 in the stadium præatacticum, 6 in the stadium atacticum, 8 in the stadium paralyticum), in 25 cases it was bilateral (3 in the stadium præatacticum, 3 in the stadium atacticum, 19 in the stadium paralyticum).

Myosis was found with unilateral sympathetic lid paralysis 6 times, with bilateral 11 times. *In general this slight drooping of the upper lid increases from the beginning of the tabes up to the stadium paralyticum.*

The palpebral fissure may appear of normal width, and yet a sympathetic ptosis exist, since in tabes the lower lid is often relaxed and drooping. It is best to make the examination for sympathetic ptosis when the line of vision is horizontal. In the normal relation, the upper lid hangs over the upper corneo-scleral margin or perhaps over the middle of the upper iris diameter (the pupil being medium-sized).

When the edge of the upper lid stands at the height of the pupillary margin or partly covers the pupil, there can be no doubt as to the existence of a sympathetic ptosis of the upper lid.

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[1] Jacobson, " Augenaffectionen bei Allgemeinleiden," p. 64.

Closure of the Lids.

If one causes a tabic patient to close the eyelids, after some seconds *fibrillar twitching is seen in the musculus orbicularis palpebrarum.* If only one eye be closed, the twitching is seen also in the orbicularis of the other side. This twitching is the more violent, the greater the effort that is required to close the lids.

This fibrillar twitching is known to exist in other nervous diseases. It is very evident, for example, in hysterical patients in the hypnotic state.

As I learn from Dr. Zieminski of Warsaw, it seems to be known in tabes, although I do not find it described in the literature. My friend, Dr. Müller, at my suggestion, has given his attention to this symptom in Prof. Krafft-Ebing's clinic in Graz. He has verified my observations, yet found the twitching, although weak, in apparently healthy persons.

I believe that the reason why this orbicularis twitching is so striking in tabic patients is that in tabes the closure of the lids is difficult, a fact to which Prof. Déjerine called my attention. It often happens that the corrugator supercilii, or other cutaneous muscles in the neighborhood of the eye, must be called into play to accomplish the closure.

This difficulty of closure of the lids in tabes would seem to me to indicate a slight affection of the upper nucleus of the facial nerve. We shall show later that paralysis of the facial nerve occurs in tabes and is probably of central origin. That only the upper twigs of the facial nerve should be so frequently attacked, may depend on the fact that, according to Mendel,[1] these twigs spring from a separate nucleus, which is in close relation with that of the oculomotor nerve, a nucleus also frequently affected in tabes.

Epiphora in Tabic Patients.

I have seen this unilateral once each in the stad. præatact., atact., and paralyt.; most frequently it is bilateral. I found epiphora in 50 cases altogether, *i.e.*, in about half the patients. It was found bilateral 9 times in the stad. præatact., 23 times in the stad. atact., 15 times in the stad.

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[1] Mendel, *Berliner klin. Wochenschr.*, 1887, No. 48.

paralyt. The epiphora increases in frequency then from the beginning of the tabes up to the stad. atact.; in the stad. paralyt. it is relatively about as frequent as in the stad. atact.

For the literature of epiphora in tabes, I would refer to the paper by Petrolacci.[1]

In connection with the epiphora stands the frequently observed injection of the conjunctiva, the occurrence of blepharo-adenitis, especially at the outer canthus, and the less frequent eversion of the lower punctum.

The epiphora is probably in part due to the difficulty in the carrying off of the tears (weakness of the orbicularis); it is due in greater part, however, to *hypersecretion.* The latter has its analogy in the hyperidrosis of the face (Remack, Althaus), an accompanying symptom of the vasomotor neuroses in tabes. Claude Bernard observed after cutting the sympathetic an increased perspiration of the same side.

The Intraocular Tension.

Hitherto no attention has been given to the condition of the tension in tabes, although on theoretic grounds we would suppose that nervous affections might influence it. Disregarding two cases where a diminished tension existed in the beginning of the tabes, I found a number of times the tension so diminished that the ball was soft. The hypotonia was found:

	In the stadium			
	præatact.	atact.	paralyt.	with myosis.
On one side..............	2	11	11	9
On both sides............	4	4	4	3

Thus altogether hypotonia existed in 36 cases, and in 12 cases (= $\frac{1}{3}$) was accompanied by myosis.

The nerve fibres which regulate the intraocular tension are in the sympathetic nerve.[2] According to the investigations of Hippel and Grünhagen,[3] only very few of the fibres lie in the upper portion of the cervical sympathetic, the greater part pass into the sympathetic at the level of the upper cervical ganglion. Section of the sympathetic is known to cause a diminished tension, irritation of the central stump of the sympathetic, an increased tension with contraction of the vessels (Adamük[1]).

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[1] Petrolacci, De l'epiphora ataxique, *Thèse de Montpellier*, 1886.

[2] *Cf.* the well-known experiments of Claude Bernard.

[3] Hippel and Grünhagen, *Graefe's Archiv*, vol. xvi., 1, p. 39.

Since the intraocular tension is but a quota of the vascular pressure, it may be supposed that the latter is diminished in tabes.

This agrees with the experimental studies of Ludwig and Thiry, who found, after cutting the medulla between the first and second cervical vertebræ, a diminution of vascular tension.

Clinical observation has shown that the intraocular tension is independent of the width of the pupil (Schnabel[2]).

Schnabel can understand the relaxation of the iris vessels and the simultaneous existence of diminished tension only on the supposition that the other vessels of the uveal tract, especially those of the ciliary body, are contracted.[3]

The Pupil.

Width of the Pupil.—A frequent symptom in tabes is an inequality in the size of the pupils. Charcot[4] especially has lately called attention to this symptom in his paper on the visual disturbances in tabes. Rehlen,[5] however, claims to have found inequality of the pupils only twice in 35 cases of tabes.

I found it 32 times—*i.e.*, in 27.6 % of the cases examined. I have, moreover, found that *there is no marked difference in the relative frequency of inequality of the pupils in the different stages of tabes; it is but slightly more frequent in the initial stages.*

Pupils of equal size were found in 72 cases (in 7 cases, on account of synechiæ, etc., no significance could be attached to the size of the pupils) with

		In the stadium		
		præatact.	atact.	paralyt.
Mydriasis	20	7	5	8
Myosis	26	6	13	7
Medium-sized pupil	26	6	12	8

From this tabulation it may be seen that mydriasis is of more frequent occurrence in the beginning of tabes than in the stadium atact., and again becomes more frequent in the stadium paralyt. Myosis occurs most frequently in the stadium atact.

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[1] Adamük, *Centralbl. f. d. med. Wissensch.*, 1867, No. 28, p. 433.

[2] Schnabel, *Arch. f. Augenheilk.*, vol. xv., p. 330.

[3] Schnabel, *l. c.*, p. 331.

[4] Charcot, *Rec. d'Ophtal.*, Nov., 1887.

[5] Rehlen, *Aerztl. Intelligenzbl.*, Nos. 11–14, 1882.

Form of the Pupil.—In regard to this point, I must call attention to an irregularity of the pupil hitherto unnoticed. In tabes the pupil is often elliptical, with its long diameter mostly oblique from downward and outward to upward and inward, and symmetrical in the two eyes (14 cases), less frequently the long diameter is vertical (11 cases), or has some other direction. Altogether I have found in 32 cases a marked deviation of the pupil from the circular form. Seven cases were in the stadium præatact., 20 in the stadium atact., and 5 in the stadium paralyt.; in nearly every case myosis existed.

Explanation of the Spinal Myosis.—I believe that this irregularity of form disproves the supposition that the spinal myosis is caused by a spastic contraction of the sphincter pupillæ; it is caused, on the contrary, by a paralysis of the iris vessels, varying in degree in different meridians.[1] It would be difficult to believe in tabes, where paralysis of the accommodation often accompanies the myosis, that of two intraocular muscles whose nerve nuclei lie close together, and whose nerve fibres run in company to the anterior portion of the eyeball, one muscle should be for years in a state of spasm and the other in a condition of paralysis.

On purely theoretic grounds Heddaeus[2] stated, without having seen such cases, that irregularity of form of the iris would indicate anomalies in the centrifugal pupillary fibres (inclusive of their terminations in the iris).

Hensen and Völckers[3] have observed a pear-shaped pupil dependent on paralysis of a single ciliary nerve.

The *myosis paralytica* is characterized by the fact that the mydriasis after atropine may last from four to five weeks, while in myosis spastica it is of short duration. After eserine and pilocarpine the pupil becomes slightly smaller.

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[1] Berger, E., Recherches sur les troubles oculaires dans le tabès dorsal, *Compt. rend. de l'Académie des Sciences*, Paris, June 4.

[2] Heddaeus, "Die Pupillarreaction auf Licht," 1886, p. 58.

[3] Hensen and Völckers, "Untersuch. über den Mechan. der Accom.," Kiel, 1868.

According to Biffi, Cramer, and De Ruiter, atropine irritates the sympathetic and paralyzes at the same time the oculomotor nerve. The action of atropine on the pupil with myosis paralytica can then be explained only in the latter manner.

We know to-day, after the investigations of Grünhagen, Eversbusch, and others, that a proper dilatator pupillæ does not exist, but that the antagonists of the sphincter pupillæ are the vaso-constrictors of the iris.

Claude Bernard[1] accepts the existence of vaso-constrictors of the iris, and also of fibres which innervate the dilatator pupillæ, and believes that they arise from various parts of the cord.

According to Bridge these fibres have their origin in a cilio-spinal centre in the cord.

According to Salkowski,[2] the vasomotor and pupil-dilating fibres have their centre in the *medulla oblongata*.

Schiff has seen, after section of a lateral half of the medulla, dilatation of the iris vessels of the same side. According to Snellen, Hippel, and Grünhagen, and Leber as well, the pupil-dilating fibres run with the fibres of the fifth nerve; according to Balogh, they all enter the ganglion of Gasser.

Leeser[3] believes that the pupil-dilating fibres spring from the medulla, the cervical portion of the cord, and the cervical sympathetic.

As an explanation for the spinal myosis, Förster[4] assumed an affection of the cilio-spinal centre of Bridge (situated in the lower cervical and upper dorsal portions of the cord). Hempel[5] wished to explain it by an affection of the centre for the pupillary reflex.

J. Hutchinson, Jr., states that myosis can occur in inflammatory affections of the brain, even when the affection is of a part distant from the corpora quadrigemina.

Rählmann[6] noticed vasomotor paralyses of the same side of the face on which a myosis existed; Gowers saw hyperidrosis of the same side.

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[1] Claude Bernard, *Compt. rend. de l'Acad. des Sciences*, 1862, vol. iv., p. 382.

[2] Salkowski, *Zeitschr. f. rationelle Med.*, vol. xxix., pp. 167–190.

[3] Leeser, "Die Pupillarbewegung," Wiesbaden, 1881, p. 29.

[4] Förster, *Graefe and Saemisch's Handbook*, vol. vii., p. 134.

[5] Hempel, Uber die Spinalmyosis, *Graefe's Archiv*, 1876.

[6] Rählmann, *Sammlung klin. Vorträge*, No. 135, p. 8.

I may mention here that I have succeeded in producing myosis in dogs, experimentally, by wounding the upper portion of the fossa rhomboidalis. It is questionable whether in tabes the paralysis of the vaso-constrictors is caused by affection of the centre in the medulla for the vaso-constrictors of the iris, or in a reflex manner from the cord. The former is more probable.

A *spastic myosis* is also supposed to occur (? in tabes).

Hempel[1] grants the possibility of a myosis spastica from a secondary contracture of the sphincter, with co-existing paralysis of the sphincter and of the pupil-dilating fibres. Michel[2] says spastic myosis occurs after hemorrhage into the ventricles.

In place of the paralytic myosis, transitory dilatation of the pupil may occur during the gastric crises or the lancinating pains, as Duchenne de Boulogne[3] first observed.

A spastic mydriasis, which may be produced by irritation of the posterior columns, can be of long duration in the initial stage of tabes. It is incomprehensible why its occurrence in tabes has been denied by so many authors. A more or less marked mydriasis seems often to precede the spinal myosis.

Chauveau produced dilatation of the pupil, experimentally, by irritating the posterior roots of the cord.

Schiff and Foa were able to obtain a reflex dilatation of the pupil from the cord, but only from its posterior columns.

The irritation of the respiratory centre in the medulla is also followed, as Schiff has shown, by dilatation of the pupil. It is probable that here the vasomotor centre for the iris vessels is irritated at the same time.

Paralytic mydriasis is at times observed in conjunction with paralysis of the accommodation, as one of the earliest symptoms of tabes. I have seen it occur twice unilaterally, and seven times bilaterally, in the stadium præatact.

It is striking that in paralytic mydriasis, contrary to the spastic form, atropine does not produce so much dilatation of the pupil as it does in the normal eye.

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[1] Hempel, *Graefe's Archiv*, vol. xxii., 1, pp. 1–28.

[2] Michel, *l.c.*, p. 519.

[3] Duchenne de Boulogne, *Gaz. hebd.*, 1864, p. 147.

As a point in the differential diagnosis, Förster mentions the fact that in paralytic mydriasis the direct reaction of the pupil to light may be abolished, while the consensual reaction, as well as the reaction from convergence, may be preserved.

The Pupillary Reaction to Light and Accommodation.

The behavior of the pupil under the stimulus of light and accommodation is of great importance in the diagnosis of tabes. The reaction of the pupil to light is lost first, later the reaction to accommodation. This peculiarity is found also in progressive paralysis, otherwise it is rare. According to Stolzenberg (1883), the irresponsive pupil is found in many cases of cerebral syphilis. Uhthoff[1] found it in one case of bisulphide of carbon poisoning, together with myosis and retrobulbar neuritis. I found it as follows:

	In the stadium		
	præatact.	atact.	paralyt.
Good reaction of the pupil	3	1	0
Gowers' symptom	6	2	0
Slow light, good accommodation reaction	2	1	2
Argyll-Robertson symptom, unilateral . .	0	2	0
" " " bilateral . .	10	24	18
Pupil irresponsive to light and accommodation, unilateral	0	1	2
Pupil irresponsive to light and accommodation, bilateral	0	11	10

This table shows that good pupillary reaction was found in four cases only, *i.e.*, in 3.7 % of the patients, and only in one case in which the ataxy was well marked. In eight cases reaction to light was preserved, but the pupillary contraction was of short duration.

After a marked contraction of the pupil a dilatation followed, and then some vacillation. This sort of pupillary contraction in tabes was first described by Gowers (1883). I shall therefore call it Gowers' symptom.[1]

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[1] Uhthoff, *Graefe's Archiv*, vol. xxxiii., 1.

Three fourths of the patients who presented this symptom were in the stadium præatacticum, one fourth in the stadium atacticum.

Five patients with preserved accommodative reaction showed only a slight light reaction; two of these were in the stad. præatact., one in the stad. atact., and two in the stad. paralyt.

This shows, then, that a *trace of pupillary reaction to light may be preserved for a long time.* Statistics as to the relative frequency of failure of the pupillary light reflex and of the patellar tendon reflex have not been made. Moeli[2] found, however, that the irresponsive pupil is found more frequently in conjunction with Westphahl's symptom than with preserved knee-jerk.

In two cases the reaction to light was wanting on one side and preserved on the other.

Both patients were in the stad. atact. In one case the irresponsive pupil contracted when light was thrown into the other eye. This observation accords with theoretic arguments. Leeser[3] says that the fibres producing pupillary reaction run in tracts separate from those fibres producing consensual reaction.

In 52 cases the pupils were absolutely irresponsive to light, while they contracted during the act of accommodation. The Argyll-Robertson symptom was found present 10 times in the stad. præatact., 24 times in the stad. atact., and 18 times in the stad. paralyt.

In 21 cases the pupils of both eyes were absolutely irresponsive to light and to accommodation; 10 of these were in the stad. atact., 11 in the stad. paralyt.

We conclude from these statistics that in tabes the reaction of the pupil is gradually lost, first in response to light, and later in response to accommodation, and that with few exceptions the reflex paralysis of the pupils of both eyes occurs simultaneously. Gowers found a unilateral irresponsive pupil only once in 72 cases.

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[1] It should, however, be remembered that rythmic oscillation of the pupil under the stimulus of light occurs in multiple cerebral sclerosis, after epileptoid attacks and in hysterical convulsions.

[2] Moeli, *Centralbl. f. Augenheilk.*, 1887, p. 179.

[3] Leeser, *l.c.*, p. 13.

From the fact that a period of increased patellar reflex precedes the failure of the reflex, we might assume that a period of increased pupillary reaction to light precedes the non-reaction. Parinaud[1] has observed this in three cases of disseminated sclerosis.

As is well known the accommodative pupillary reaction may be tested in amaurotic patients by having them fix their own hand. *The reaction of the pupil to light disappears undoubtedly earlier than the reaction to irritation of the skin.* The latter may be well preserved for a long time. Gentle pressure on one eyeball may cause dilatation of the pupil of the other eye.

As to the frequency of the reflex iridoplegia, Moeli found it in progressive paralysis in 47 % of the cases, in 4 % it was doubtful, in 10 % there was slight, in 10 % sluggish, in 28 % good, light reaction. It is a valuable point in the diagnosis of progressive paralysis, since, according to Moeli,[2] it appears in only 1.4 % of the non-paralytic insane. Erb believes disturbances of the pupil and of the accommodation to exist in about half the tabic patients. This estimate, in comparison with my figures, is too small, since I found the Argyll-Robertson symptom alone in nearly half my patients. Gowers found the reflex iridoplegia 48 times in 72 tabic patients, Vincent[3] 47 times in 50 patients (25 times with myosis, 6 times with mydriasis, and 11 times with a medium-sized pupil).

In my cases of reflex iridoplegia there was:

Mydriasis	in	11	cases.
Myosis	"	24	"
Medium-sized pupil	"	15	"
Myosis on one side and mydriasis on the other,	"	2	"

To explain the phenomenon of the immobile pupil we must understand the anatomical course of the nerve filaments that produce the light reaction.

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[1] Parinaud, *Rec. d Ophtal.*, 1882, p. 688.

[2] Moeli, *Neurolog. Centralbl.*, 1885, p. 354.

[3] Vincent, Des phenom. oculo-pupil. dans l'ataxie locom., *Thèse de Paris*, 1877.

We know that these fibres are found only in the centre of the retina, and that they run in the temporal side of the optic nerve together with the macular fibres. According to Gudden they are recognizable anatomically as thicker fibres. They are generally resistant to diffuse pathological processes, as cases prove, in which the light reflex of the pupil was preserved in amaurotic eyes (Haddaeus).

These fibres run in the temporal side of the optic nerve to the tract, which the condition in hemiopia would indicate. The right or left-sided hemiopia is associated in many cases with a dilatation of the pupil of the same side.[1]

From this it follows that the nerve fibres going to the nucleus of the oculomotor nerve, are contained in the same bundles as those nerve fibres (temporal) which belong to the paralyzed half of the retina.

No direct investigations have been made concerning the further course of these fibres. Knapp[2] found contraction of the pupil with preserved light reflex after cutting the cord between the fifth and sixth cervical vertebræ, the centre for the light reflex therefore lying above the fifth cervical vertebra.

Meynert found fibre connections between the nucleus of the oculomotor nerve and that part of the optic nerve which runs through the corpus geniculatum internum and the brachium conjunctivum anticum to the gray matter of the corpora quadrigemina.

A pathological condition found by Moeli[3] has thrown light on the further course of the fibres producing the light reflex. In a case of a pupil irresponsive to light and normal optic nerves, the autopsy showed a tumor in the anterior section of the third ventricle. The optic nerves and the nucleus of the oculomotor nerves were intact. The loss of pupillary reflex was doubtless caused by injury of the pupillary reflex fibres near the ventricle wall in their course to the nucleus of the oculomotor nerve.

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[1] *Cf.* Michel, *l.c.*, p. 518.

[2] Knapp, *Arch. f. Augen- und Ohrenheilk.*, vol. ii., 2, pp. 191–196.

[3] Moeli, *Arch. f. Psych.*, vol. xviii., 1887, p. 1.

Argyll-Robertson[1] explained the symptom which he described, by supposing that the cilio-spinal fibres formed a part of the connecting chain between the retina and the pupil, and that the reaction of the pupil did not arise as reflex activity, but as a periodic interruption, this latter failing in tabes.

Since the experiments of Knapp and the investigations of Moeli, we can no longer doubt the correctness of Mauthner's[2] explanation of the immobility of the pupil in tabes, namely, that **the fibres for the pupillary light reflex, lying in the wall of the third ventricle, suffer from a chronic ependymitis and a consequent sclerosis.**

Paralysis of the Accommodation.

Galezowski reported to the Société de Biologie (February 18, 1882) several cases of tabes in which a paralysis of accommodation was found, together with normal pupils and loss of sensation in the skin of the orbital region; the affection was most frequently unilateral.

Hirschberg remarked in a report, that a well-known oculist presented himself at von Graefe's clinic in 1867 with an isolated paralysis of accommodation, and later died of tabes.

I mentioned at the same meeting of the Société de Biologie similar cases that had come under my observation. I have seen in two cases a paralysis of accommodation without paralysis of the sphincter in the beginning of tabes. In these cases, however, there was already a reflex iridoplegia.

I have seen such cases more frequently in the later stages of tabes (23 times in 109 cases). I have also found an extensive anæsthesia of the fifth nerve early in the disease in two cases. I have found similar cases reported (*e.g.* by Althaus), but they are rare. In Ed. Meyer's clinic in Paris, I have had lately the opportunity of seeing a tabic patient with anæsthesia of the skin in the orbital region.

In summing up the total number of cases of paralysis or of marked loss of accommodation, I find 34 (31 %).

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[1] Argyll-Robertson, *Edinburgh Med. Journ.*, 1869.

[2] Mauthner, *l.c.*, p. 353.

We find that *the paralysis of accommodation becomes the more frequent, the more the disease is advanced; in one third of the cases only, was the paralysis of accommodation accompanied by mydriasis.*

It is not easy to understand why so little attention has been paid hitherto to the frequent occurrence of paralysis of accommodation in tabes. Erb[1] alone says that in general this symptom is not rare, it being present in about one third of his cases. Gowers, however, found paralysis of accommodation and loss of pupillary reflex only 6 times in 72 patients.

The credit of having demonstrated that the simultaneous existence of paralytic mydriasis and paralysis of accommodation is to be referred to a nuclear disturbance, is due to Parinaud (1880). Those cases, however, in which the paralysis of accommodation is complicated with paralytic myosis, are also to be explained as of nuclear origin, since this myosis, as we have seen, is caused by functional disturbance of certain regions in the medulla oblongata.

THE OPTIC NERVE.

Frequency, Commencement, and Course of the Atrophy of the Optic Nerve.

I found amblyopia with ophthalmoscopically normal fundi in 7 cases, marked atrophy of the optic nerve in 44 cases, *i.e.*, altogether 51 cases of functional disturbance of the optic nerve (46.7 %).

The estimation of the frequency of optic atrophy in tabes is very different among the different authors, being generally reckoned too high by the ophthalmologists, too low by the neurologists. Statistics on this point, as well as on a number of other points in tabes, are desirable.

If I combine my figures with those of Erb (3 cases of optic atrophy in 70 patients), Topinard (49:102) and Cyon (60:203), and average the total, I find 33.7 % of optic atrophies. Leber estimates it to be 26 %, Michel 12–13 %, Althaus $\frac{1}{6}$ of the cases, Moeli[2] 13.5%.

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[1] Erb, *l.c.*, p. 179.

[2] Moeli, *Arch. f. Psychiat.*, vol. xiii.

As to the period when optic atrophy develops, the general statements are that it is mostly in the beginning of tabes; some authors, as Schmeichler,[1] wrongly deny that optic atrophy can develop with marked ataxy.

In seven cases of tabes the optic atrophy was the first symptom and spinal symptoms appeared later.

In one case nearly two years elapsed between the optic atrophy and the appearance of spinal symptoms (lancinating pains).

Yet, there are cases in the literature of much greater interval between the affection of the optic nerve and that of the cord, as one case of Charcot's of ten years' interval, one case of Förster's[2] of three years' interval, and some cases of Gowers[3] of fifteen to twenty years' interval.

The optic atrophy frequently precedes the paralyses of the ocular muscles, as Michel[4] correctly states. *In general, paralyses of the exterior ocular muscles are found disproportionately oftener in cases showing optic atrophy than in cases uncomplicated with optic atrophy.*

	Muscle paralyses.	No muscle paralyses.
With amblyopia . .	4	3
" optic atrophy .	25	19
" normal optic nerves	23	32

The cases with paralyses of the ocular muscles are more disposed to optic atrophy than the cases without paralyses.

In regard to the cases in which one eye is affected with optic atrophy before the other, little is to be found in the literature. Leber[5] says that ocasionally one eye only is found to be affected and a long interval elapses before the second eye is affected, mentioning, *e.g.*, a case in which four years after the first eye had become blind the other had V = ½.

In twelve of my cases it was distinctly stated by the patient that the vision of one eye failed before that of the other. The shortest interval between the affection of the two eyes was two months, the longest ten years.

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[1] Schmeichler, *l.c.*, p. 452.
[2] Förster, *l.c.*, p. 131.
[3] Gowers, from a citation in the *Neurolog. Centralbl.*, 1883.
[4] Michel, *l.c.*, p. 628.
[5] Leber, *l.c.*, p. 866.

It was remarkable that *the left eye was first affected with optic atrophy twice as frequently as the right eye.* So also in glaucoma, the left eye is more frequently the one first to be affected. In my examinations as to the condition of the vision and the visual field, in the great majority of cases the affection was further advanced in the left eye, from which we may assume that the left was the eye first affected.

The most varied statements are found in the literature as to the *course of the optic atrophy.*

Erb[1] says indefinitely, that blindness may follow after weeks, months, or years.

Leber[2] says that, as a rule, one to two years are required for the course of the atrophy and the production of total blindness. Schmeichler[3] believes that the atrophy in tabes seldom leads to complete amaurosis, and mentions a case of atrophy which lasted thirteen years without causing total blindness.

Althaus[4] says: "La marche de l'atrophie est généralement lente et progressive discontinue."

Gowers (1883) believes that the atrophy developing early in the course of tabes, leads more quickly to blindness.

From a practical stand-point, all cases in which the vision is so poor that only fingers can be counted, may be considered blind, as Magnus has classed them in his statistics of blindness. The quickest development of blindness was 2 months, in a syphilitic patient. The slowest development in a syphilitic patient was 15 years, in a non-syphilitic patient, 17 years.

The average period of development of blindness in cases of optic atrophy complicated with ocular paralyses was 3.4 years; in cases without ocular paralyses, 3 years.

If I may be allowed to draw general conclusions from my statistics as to the course of the optic atrophy, they would be as follows:

1. *Between the cases of optic atrophy with ocular-muscle*

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[1] Erb, *l.c.*, p. 147.
[2] Leber, *l.c.*, p. 876.
[3] Schmeichler, *l.c.*, p. 457.
[4] Althaus, *l.c.*, p. 169.

paralyses and those without, there is no special difference as regards the duration of the affection before blindness ensues.

2. *The most of the optic atrophies develop in the stad. prœatact. If this stage be once passed, the danger of optic atrophy is less. Should an optic atrophy develop later, blindness will follow the sooner the more advanced is the tabes.*

3. The course of the optic atrophy seems somewhat slower in syphilitic patients than in non-syphilitic.

Subjective Symptoms.—The subjective symptoms in the beginning of tabes are, as is well known, an increased sensibility to light, and the seeing of sparks and muscæ volitantes.

In the beginning, and at times later in the course of optic atrophy, subjective color-sensations come on. Leber[1] reported one case in which an intense red glimmer appeared in the defective part of the visual field. In another case of Leber's a yellowish cloud was seen in the visual field, which became bluish-gray as it vanished.

I have seen *erythropsia* twice in tabes. In one case it came on after strychnine injection, but the patient had at the same time zoöpsia, from the abuse of alcohol. In the second case, also in a female, the attacks of erythropsia came on in the evening, and began on the temporal side.

The first case shows that the sensation of red does not always depend on a hyperæsthesia of the retina for red—this was not once found,—but that it has in a number of cases a cerebral cause.

Visual hallucinations are rare in tabes. In such cases we should suspect a complicating psychic affection.

The Central Color-Perception.—The disturbances of color perception in tabes have, as is well known, no direct connection with the loss of vision and the contraction of the visual field. When a disturbed color-sense is found in connection with a normal or increased power of perceiving a minimum amount of light, if a decrease in the power of distinguishing different intensities of illumination should also be found, we are at liberty, even with an ophthalmoscopically normal fundus, to diagnose optic atrophy.

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[1] Leber, *l.c.*, p. 866.

Leber and Jacobson[1] find that in the optic atrophy of tabes the perception of green fails first. Erb[2] gave the following order for the loss of color-perceptions: (1) green, (2) red, (3) yellow, (4) blue; yet he grants that there may be deviations from this order.

In 48 cases of optic atrophy I found the central color-perception preserved three times with atrophic disc, four times with normal fundus.

In 3 cases the achromatopsia began with green blindness, and in 1 case with red blindness.

In 2 cases there was red and green blindness.

In 5 cases only blue was recognized.

In the other cases no colors were recognized.

Limits of the Visual Field.—As to the co-existence of ophthalmoscopic changes, contractions of the visual field, and of the fields for colors, I have found the following:

1. *Ophthalmoscopically normal* . . . 7 cases.

Of these:

(*a*) Normal vision, normal central color-perception and contraction of the field of vision . 3 "

(*b*) Lessened vision, normal central color-perception and contraction of the field of vision 2 "

(*c*) In one case the field of vision, the central color-perception, and the vision were normal, the color-fields contracted. The other eye showed sector-formed defects of the field of vision and green-blindness.

(*d*) In one case the vision was lessened, and achromatopsia for green and red existed with an ophthalmoscopically normal fundus.

In two cases the field of vision of one eye was normal, of the other contracted.

2. *Atrophy showing ophthalmoscopic changes* existed three times with good central color-perception, of which one with normal field of vision, but lessened acuity.

In one case the central color-perception and vision were normal, the field of vision and the color-fields contracted.

In all the other cases the vision, color perception, and limits of visual and color-fields were simultaneously affected (8 cases, not including the cases of very poor vision or amaurosis).

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[1] Jacobson, *l.c.*, p. 54.
[2] Erb, *l.c.*, p. 181.

From this we may conclude, that *no relation exists between the contraction of the visual field, the color-fields, the disturbances of central vision, and of color-perception in tabes*, wherein I agree with Leber[1] and Michel.[2]

Undoubtedly, however, contraction of the visual field precedes affection of the color-sense (Leber, Schön[3]).

The form of contraction of the visual field is described in the literature in the most contradictory manners.

Graefe[4] found that the defect in the visual field begins mostly on the nasal side, Schweigger that it begins oftener on the temporal side. Förster[5] says that the contractions are mostly upward and outward, or outward; according to Leber,[6] they are mostly concentric. Erb finds that the field is progressively narrowed, at times upward and outward, at times more outward. Schmeichler[7] says: "The characteristic field of vision is the concentrically contracted. In tabes the optic nerve is uniformly (?) affected by the atrophic process, the peripheral portions of the retina becoming at the commencement of the disease incapable of performing their function." According to Galezowski, the atrophy shows in the beginning, especially in syphilitic patients, a peripheral contraction of the field.

From the variety of the statements in regard to the characteristic field in cases of optic atrophy in tabes, we may conclude that in all probability there is no characteristic field.

Of my forty-four cases, only twenty-one had such vision that, from the defects in the visual field, conclusions could be drawn as to the original contractions. At the termination of the atrophy a portion of the field between the blind spot and the point of fixation is generally long preserved, independent of what contractions may have preceded.

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[1] Leber, *l.c.*, p. 864.

[2] Michel, *l.c.*, p. 627.

[3] Schön, "Die Lehre vom Gesichtsfelde," Berlin, 1874, pp. 23–49.

[4] von Graefe: *Klin. Monatssbl.*, vol. iii., p. 134.

[5] Förster, *Congrès internat. d'ophtal.*, 1867, Paris, p. 127.

[6] Leber, *l.c.*, p. 862.

[7] Schmeichler, *l.c.*, p. 458.

From these twenty-one cases I conclude that *the contraction of the outer portion of the visual field is most frequent, not making up, however, the half of the cases,* so that we cannot speak of a characteristic contraction of the visual field in tabes.

The concentric contraction of the field has, as is known, the peculiarity that it begins with reëntrant angles whose vertices are directed toward the blind spot, never toward the fixation point. The margins of the color-fields are simply cut by these sector-formed defects.

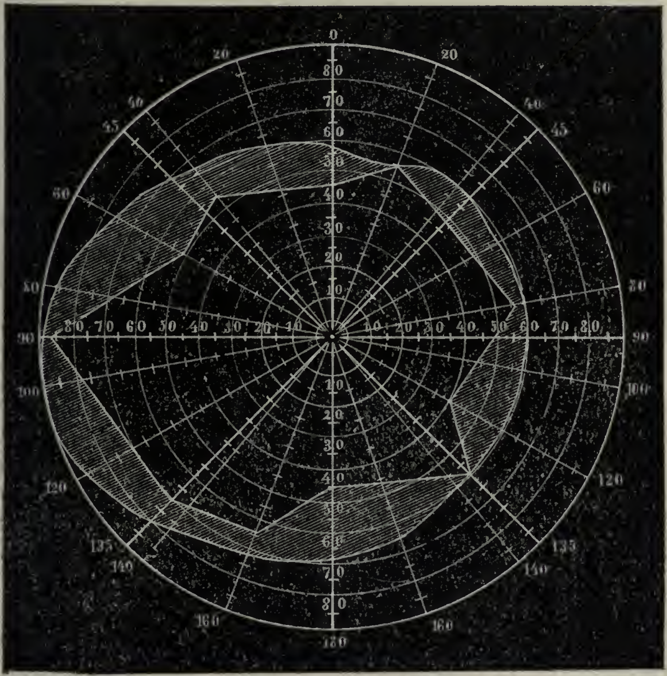

FIG. 2.—Field of vision of left eye. Jacques C.

Jacques C., æt. thirty-seven, private, had syphilis six years before. Three years before, he had diplopia, which soon disappeared. He then suffered with lancinating pains in the legs. The disease is still in the stad. præatact. The gait shows no traces of ataxy. Fatigue in walking, lancinating pains in the legs and in the thumbs, and headache in the temporal regions are the predominating symptoms of which the patient complains. His sleep is disturbed. Paræsthesia in the upper extremities. Micturition and defecation are difficult. The patellar reflex is wanting.

Bilateral paralysis of the abducens. Right, pupil of medium size; left, myosis and reflex iridoplegia. Ophthalmoscopically, normal. With movement of the eye a slight nystagmic oscillation. Left eye: $V = 1$, color-fields contracted, visual field normal. Right eye: $V = \frac{1}{10}$, color-fields markedly contracted (green 5°, red 20°, green blindness), sector-formed defects of the visual field (Fig. 2).

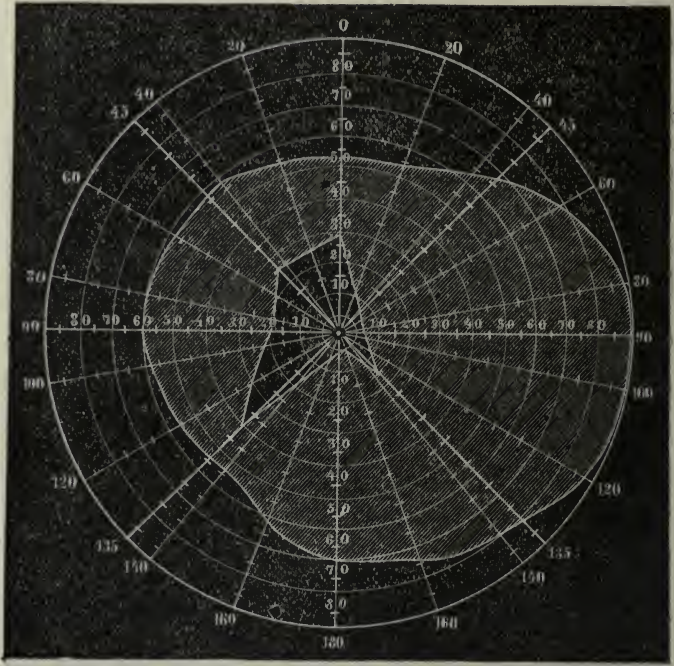

FIG. 3.—Field of vision of right eye. Alfred L.

These sector-formed defects of the visual field may appear even in very advanced cases of atrophy, and thus the visual field assume the most bizarre forms, as in the following case:

Alfred L., æt. fifty-seven, clerk, contracted syphilis when a young man. Fifteen years before, he had diplopia, which lasted forty days. Twelve years before, appeared the first spinal symptoms—fatigue in walking, lancinating pains in the legs. For three and a half years the vision has been poor. *Status presens:* pupils medium-sized; right > left, loss of light and accommodation reflexes. Right, $V = \frac{1}{8}$, blue recognized; left, movements of the hand before the eye. Ophthalmoscopically: disc pale, vessels

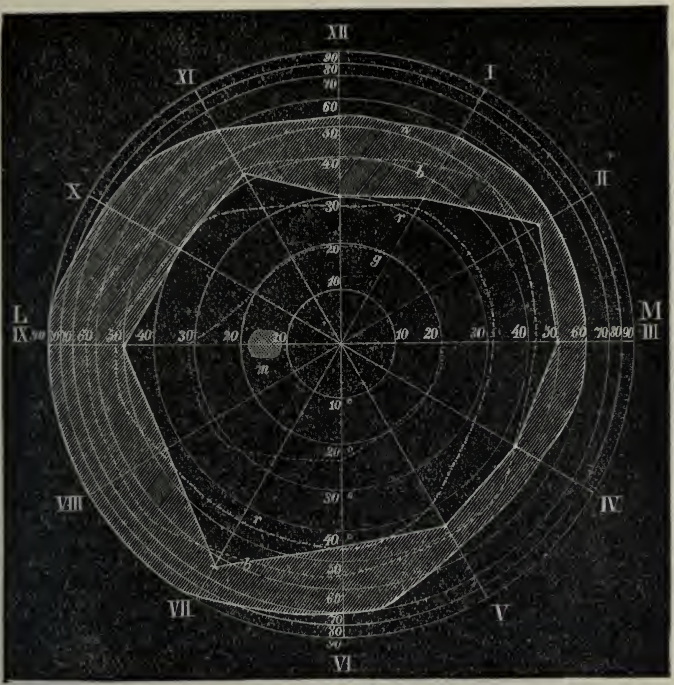

FIG. 4.—Field of vision of left eye. C.

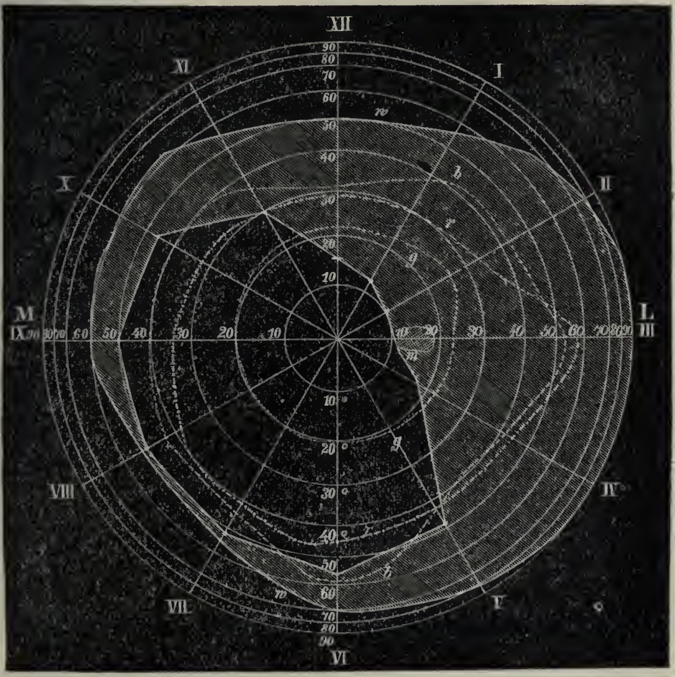

FIG. 5.—Field of vision of right eye. C.

markedly contracted. Field of vision : left, contracted at the temporal side ; right (see Fig. 3), contracted especially at the temporal side, and having a large reëntrant angle below.

The disease is in the stad. paralyt. Left : lower twigs of the facial paralyzed ; lessened sensibility of the throat. Micturition easy, defecation difficult. Sensibility of the skin of the extremities much diminished, left less than right ; patellar reflex wanting.

The contraction of the field of vision may proceed especially in a *given direction*, while in the opposite direction the field may preserve its normal limits, as may be seen in Fig. 4, from the left eye, while the right shows a narrowing at the temporal side (Fig. 5).

Ridingmaster C., æt. forty-three, consulted me in 1883. In 1886 he had caught cold at a review and suffered with "rheumatic pains." The patient had never had syphilis, yet Professor Erb prescribed an inunction treatment, which the patient did not undergo. Seven years before, lancinating pains came on. The patient connects the appearance of the first spinal symptoms with the grief which he suffered after the death of his wife. Disturbances of vision date back only a few months. On both sides, myosis and reflex iridoplegia. Ophthalmoscopically : discs pale, arteries narrow, veins normal.

Right, $V = \frac{20}{30}$; left, $V = \frac{20}{40}$. The fields of vision are shown in Figs. 4 and 5.

The following case, observed in the clinic of Professor Déjerine (Bicêtre in Paris), shows in what high degree the *field of vision may be contracted without a lessened central vision.*

Pierre M., æt. thirty-nine, cabinet-maker, contracted syphilis eighteen years before. Six years before, lancinating pains in the legs and feelings of fatigue came on. Fifteen years before, he suffered from diplopia, which lasted two years, and has not recurred. Pupils of medium size, reflex iridoplegia. Central color-perception normal. The field of vision, as Figs. 6 and 7 show, is greatly contracted. $V = 1$. Patient complains of seeing a cloud, and sees best in the evening. Both discs are uniformly pale. The disease has progressed to the stad. paralyt. The legs are paralyzed, the movements of the hands weak ; lancinating pains in the extremities. Sensibility of the skin diminished. Girdle sensations. Difficult micturition and defecation. The patellar reflexes are wanting.

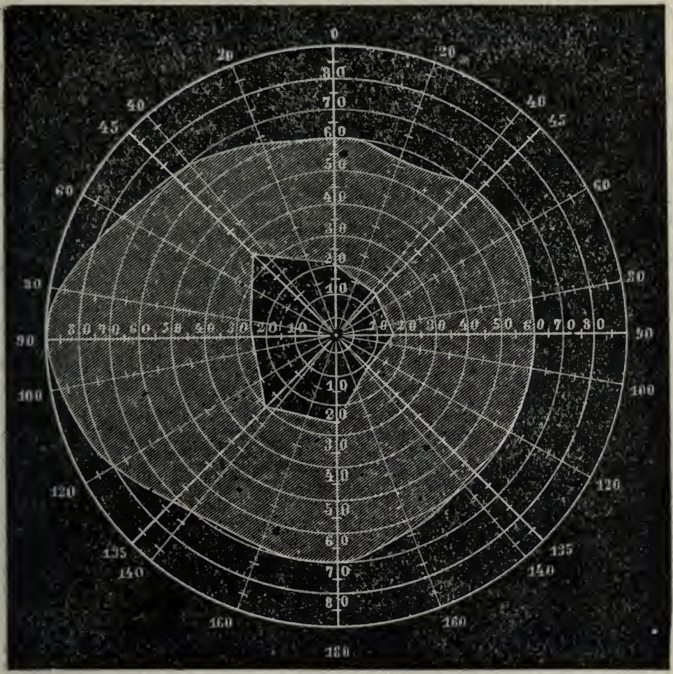

FIG 6.—Field of vision of left eye. Pierre M.

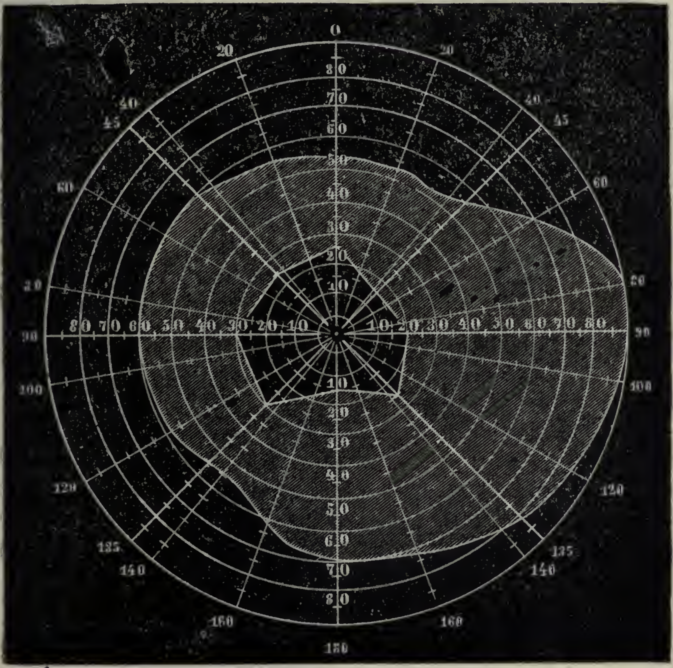

FIG. 7.—Field of vision of right eye. Pierre M.

The field of vision may contract in two opposite directions, so that it finally has the form of a cleft or of an obliquely directed oval.

I find a symmetrical contraction of the two fields very often in tabes. Most frequently the remaining portion of the field has the form of an oval with its long axis directed from upward and inward, downward and outward. Such fields are shown in Figs. 8 and 9.

These contractions of the field progressing in two opposite directions may produce a vertically oval field as in Fig. 10.

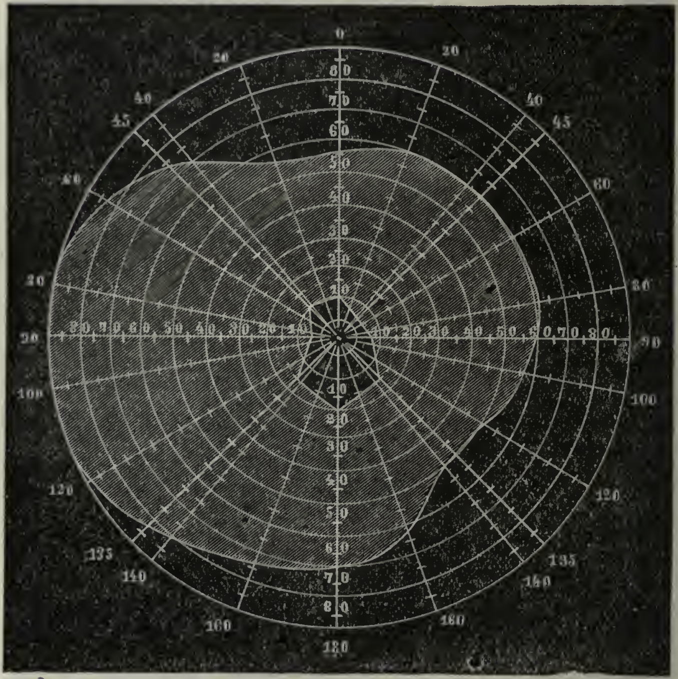

FIG. 10.—Field of vision of left eye. Leon L.

Leon L. had not had syphilis. Six years before, he found that first his left, later his right, eye tired soon in working. Both pupils are dilated and irresponsive. Left, V = fingers at $\frac{3}{4}$ *m;* right, movement of the hand before the eye. Ophthalmoscopically: optic atrophy, narrowed arteries and veins, white disc.

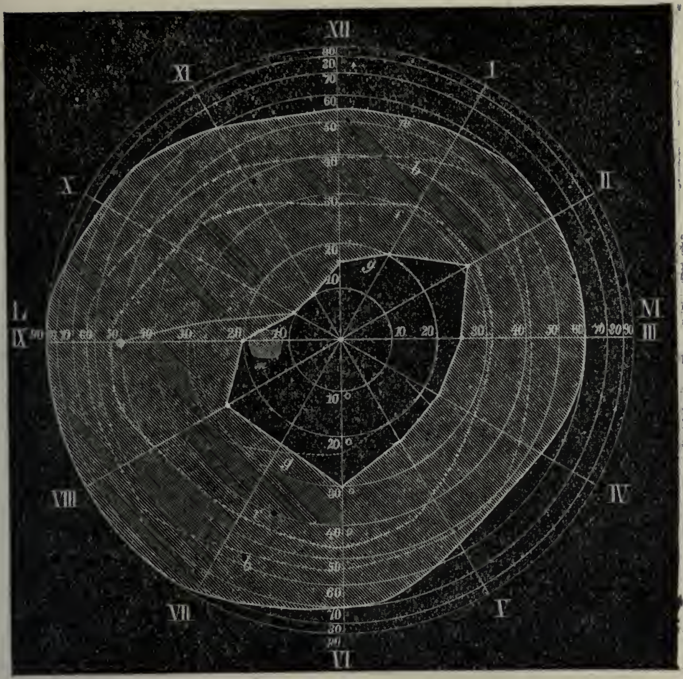

FIG. 8.—Field of vision of left eye.

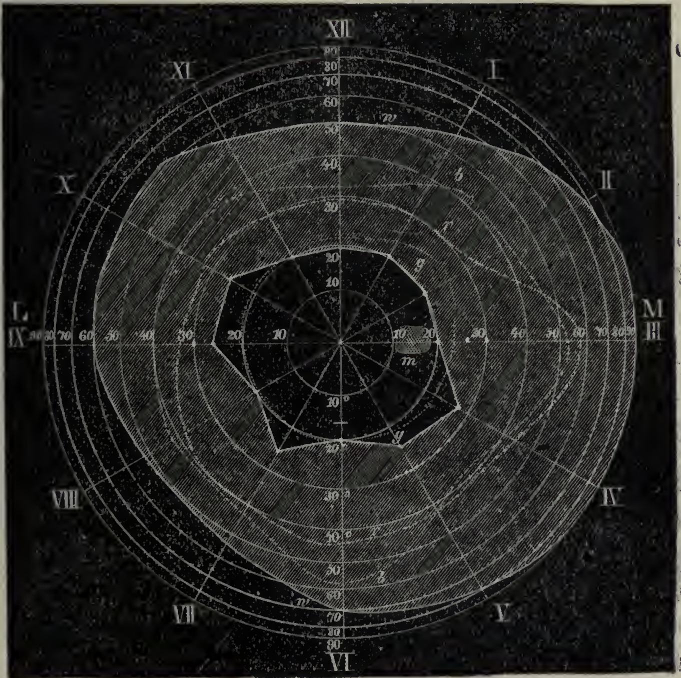

FIG. 9.—Field of vision of right eye.

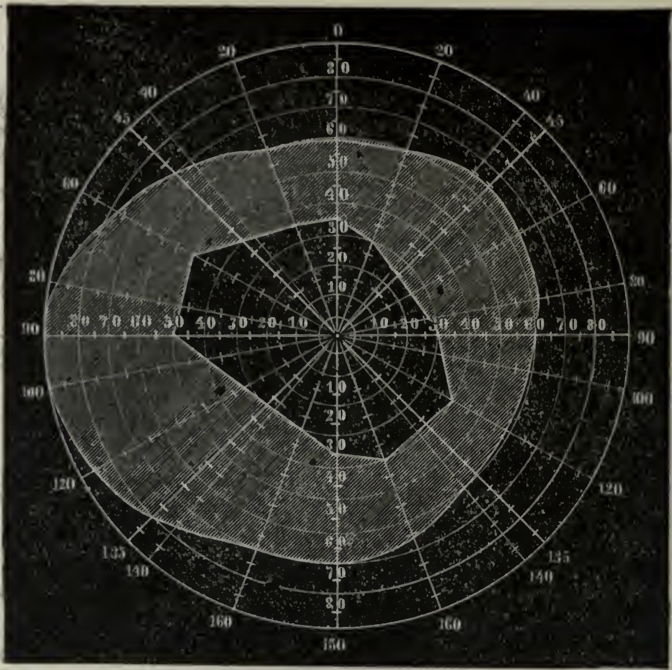

FIG. 11.—Field of vision of left eye. Florens C.

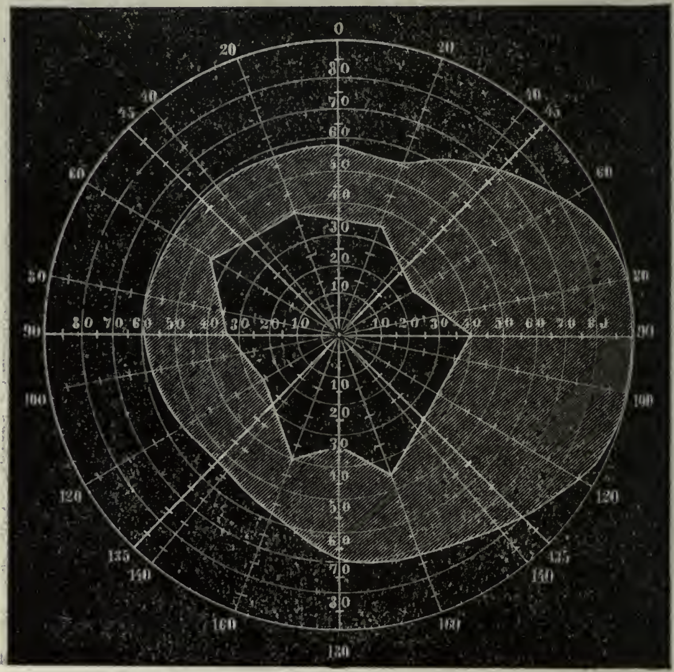

FIG. 12.—Field of vision of right eye. Florens C.

We have seen the defects of the field of vision assume a form symmetrical in both eyes; there are, however, exceptions to this. I present here a case where the defect in one field developed in a direction at right angles to that of the defect in the other eye.

Florens C., æt. fifty-four, locksmith, had not had syphilis. Ten years ago paræsthesia of the skin developed. For seven years he has suffered with disturbances of vision. The disease is in the stad. paralyt. The patellar reflexes are wanting. Romberg's symptom. Paralysis of the legs. Sensibility of the legs diminished, right more than left. Girdle sensations. Micturition difficult. Pupils of medium size, right > left, reflex iridoplegia. Right, V = fingers at 6 *m*; left, fingers at 3 *m*. Nystagmus with movement of the eyes. Ophthalmoscopically: atrophy of the optic nerves, arteries narrowed, veins dilated. Fields of vision shown in Figs. 11 and 12.

The contraction of the field of vision may also occur in its upper portion; the contraction takes place below much more rarely. Hardy[1] has described one case where the lower half of the field was completely wanting, yet this defect lasted only fourteen days and must therefore be referred to a transitory vasomotor neurosis.

I had an opportunity of seeing one case in which the contraction of the field in one eye was chiefly in the upper portion (see Fig. 14).

François Ch., æt. fifty-seven, author, had syphilis. He had shown symptoms of tabes for twenty-seven years. He had first involuntary passages, a month later lancinating pains in the legs. Fifteen years before, he suffered from diplopia, which lasted from two to three days and recurred several times. The disease is in the stad. atact. The patellar reflexes are wanting. Romberg's symptom is not present; there are lancinating pains in the hands and legs, and gastric crises. For twenty-six years he has been compelled to use convex glasses $\frac{1}{8}$ for reading (paralysis of accommodation of twenty-six years' duration!). Myosis. Central color-perception normal. V = $\frac{1}{3}$. Ophthalmoscopically, normal. Fields of vision shown in Figs. 13 and 14.

The occurrence of *hemiopia* is so rare in tabes that most authors do not mention it.

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[1] Hardy cited by Althaus, *l.c.*, p. 170.

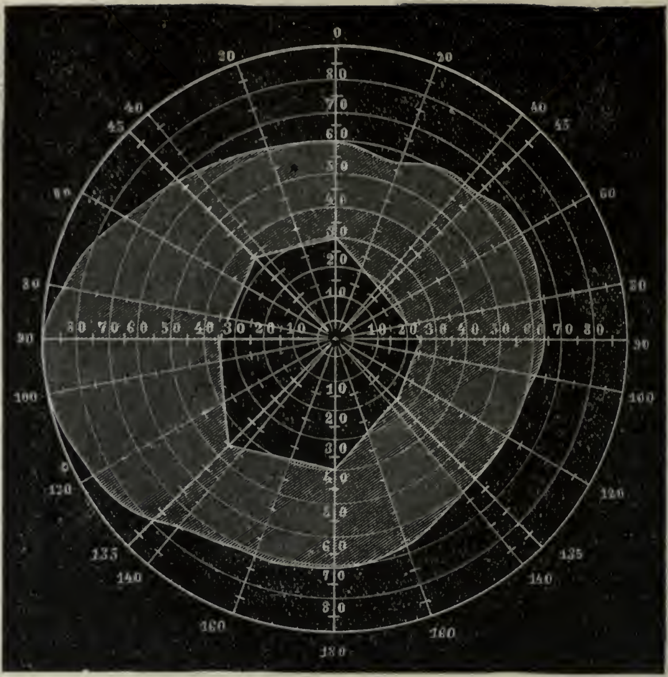

FIG. 13.—Field of vision of left eye. François Ch.

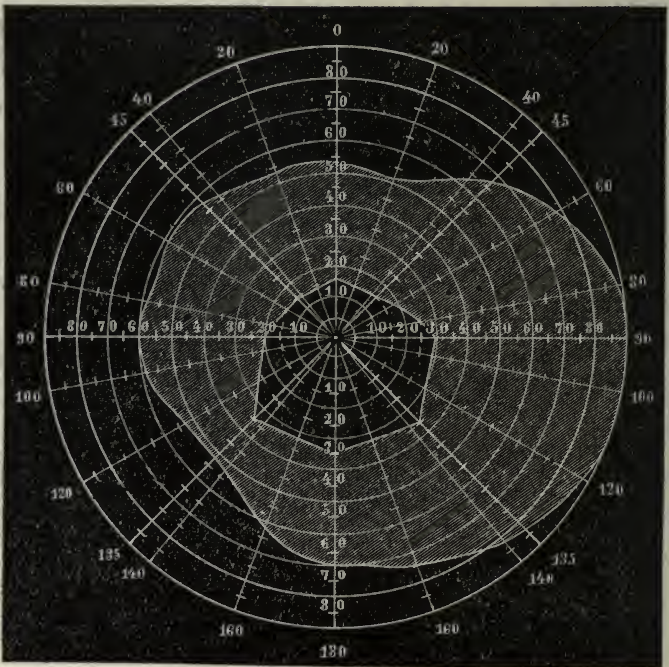

FIG. 14.—Field of vision of right eye. François Ch.

Gowers (1883) observed two cases of hemiopia in tabes, which led him to suppose a lesion of the retrobulbar optic fibres.

I have observed hemiopia in only one case, and consider it probably due to the tabic optic atrophy (Figs. 15 and 16).

The patient, Louis Adolph, had been shot in the face with a revolver. The right half of each field of vision was wanting, which would indicate a functional disturbance of the left halves of the two retinæ, and of the left optic tract. Since the revolver was placed against the right cheek, if one wishes to bring the trauma into causative connection with the hemiopia, he must suppose that the right tract remained intact and that only the left tract was injured, which is improbable, since one must at least conclude that the ball, in passing toward the tract of the opposite side, would have wounded a number of other cerebral nerves. There remains then only the supposition that this case showed a rare form of contraction of the field in tabic optic atrophy.

The occurrence of *central scotoma* in tabes is not mentioned by most authors. Leber grants the possibility of its existence in cases with poor peripheral vision. Förster[1] says, however; "According to my experience the atrophies which begin with a central defect or central scotoma do not indicate tabes. Bunge[2] reported a case of central scotoma in tabes: it was not stated, however, whether a causative connection existed between the two. Althaus[3] reported a case in which a central cloudiness was perceived in the beginning of tabes.

I have observed two cases of tabes with central scotoma.

D., æt. thirty-six, laborer, came to the clinic of Dr. Meyer, of Paris, Dec. 6, 1887. Since April, 1887, he has suffered with lancinating pains in the legs and pain in the occiput. At no time were symptoms of fatigue present. The patient has not had syphilis, but acknowledged the *abuse of alcohol.* Bilateral mydriasis, Gowers' symptom. Right, V = fingers at 3 *m*; left, V = fingers at 2½ *m.* The patient is green-blind in the right eye, and recognizes only blue with the left. As Figs. 17 and 18 show, there exists an extensive central scotoma on both sides, which in the

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[1] Förster, *l.c.*, p. 132.

[2] Bunge, "Ueber Gesichtsfeld und Faserverlauf im opt. Leitungsapparate," Halle, 1884, p. 13.

[3] Althaus, *l.c.*, p. 104.

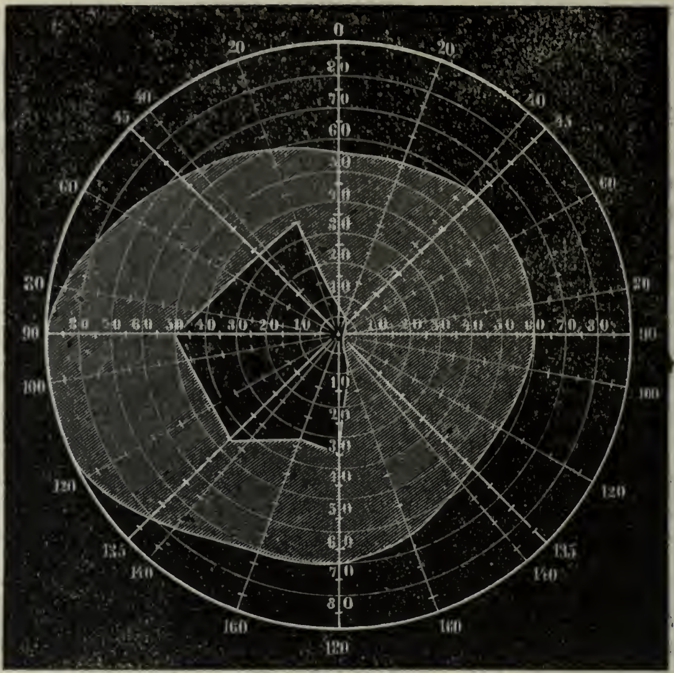

FIG. 15.—Field of vision of left eye. Louis Adolph.

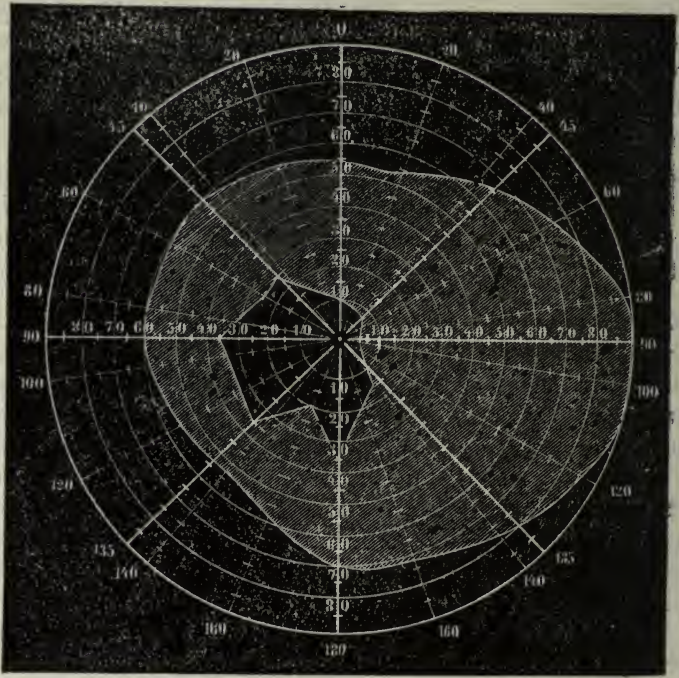

FIG. 16.—Field of vision of right eye. Louis Adolph.

right eye includes the blind spot. The dotted line gives the limits of the field for blue. Ophthalmoscopically there is a uniform atrophy with excavation of the papilla. The arteries are narrow and bordered by white lines. The veins are of normal breadth, but more tortuous than usual. The patellar reflex is abolished on the right side and weakened on the left. Micturition and defecation are normal. The throat is anæsthetic. Stad. præatact. of tabes.

Although this case showed ophthalmoscopically only the picture of uniform optic atrophy, one might suppose, since the abuse of alcohol was admitted, that the central scotoma was caused by the latter, while the peripheral contraction of the field was caused by the tabes. It is also possible for a potator having a central scotoma to become affected with tabes, and a general optic atrophy be added to the existing temporal one.

In the following case, however, there was no suspicion of the abuse of alcohol, and the peripheral contraction of the field of the right eye was undoubtedly the primary condition, and the central scotoma appeared later. This is an indisputable case of central scotoma in tabes.

B., æt. forty-eight, commander, acquired syphilis twenty years ago. He had a sunstroke two years before. From this followed a central scotoma in the left field of vision. For a month he has suffered with lancinating pains in the legs. His gait is natural. The patellar reflexes are abolished. The eyes show a nystagmus when moved.

The patient came for the first time Aug. 12, 1887. The pupils are small, left > right. The patient has epiphora. The fields of vision are shown in Figs. 19 and 20. Left : contraction of the field (30° upward, as much inward, 25° upward, 10° downward) ; the color-fields are markedly contracted ; right : the margins of the visual field are similar to those of the other side, the color-fields are a little less contracted, but a *central scotoma* exists (see Fig. 20).

When I saw the patient, Dec. 20, 1887, the vision was : right, $V = \frac{1}{10}$; left, $V = \frac{1}{20}$. In the left field also a central scotoma had developed, which, beginning at the point of fixation (Fig. 21), extended 15° toward the temporal side. The field of vision was as before, the field for blue had become reduced to the small zone in the lower portion of the field of vision, included within

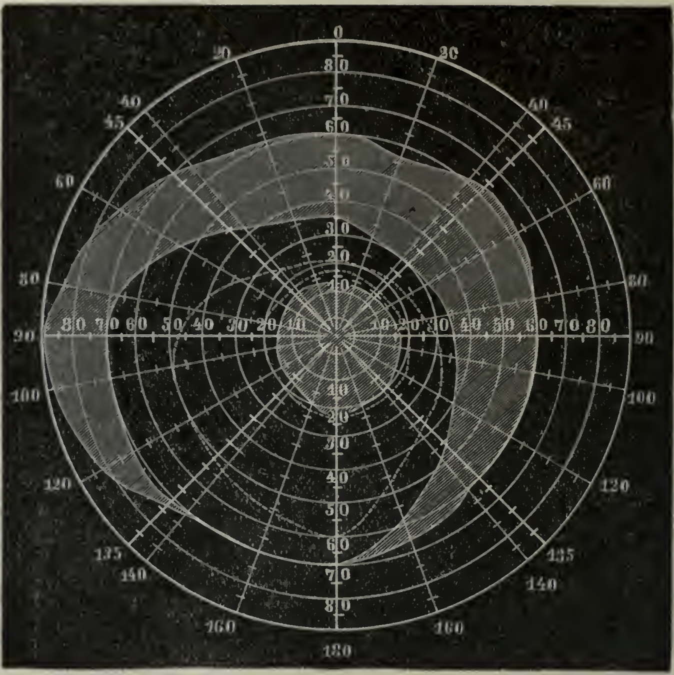

FIG. 17.—Field of vision of left eye. D.

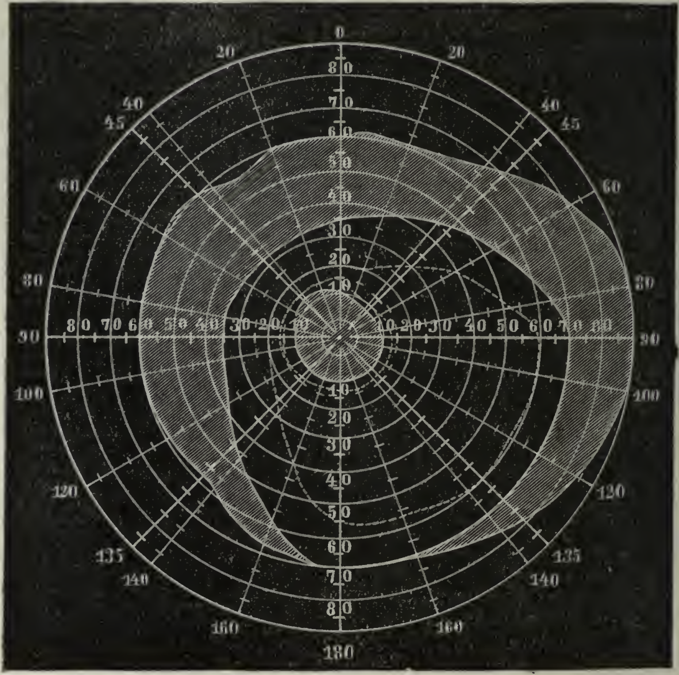

FIG 18.—Field of vision of right eye. D.

the dotted line. Other colors were not recognized. In the right eye the central scotoma had extended and had united itself with the temporal defect, so that only the nasal half of the field remained. The only color perception preserved, that for blue, was in the lower inner quadrant of the visual field (Fig. 22). Ophthalmoscopically : optic atrophy.

As to the ophthalmoscopic changes in the atrophy in tabes, a number of authors states that no stage of hyperæmia of the papilla precedes (*e.g.*, Leber[1]). Albutt[2] saw a hyperæmia precede the atrophy in a case where the latter was caused by wounds in the spine. Michel[3] describes it in the following manner: "In the beginning the papilla is more or less markedly uniformly reddened, slightly opaque, and of a grayer tone, its contours appear sharp as a rule, at the most they are slightly blurred. The vessels show no marked anomaly or are slightly overfilled."

I have had but one opportunity of studying ophthalmoscopically the beginning atrophy in tabes. The patient had noticed clouds and flashes in the visual field fourteen days before. I found a uniform redness of the papilla. The walls of the vessels were recognizable by contrast as fine stripes near the blood columns. The redness of the papilla extended to the scleral ring. The central vessels showed a normal fulness.

In beginning atrophy the nasal half of the papilla, usually redder than the temporal, appears of a uniform gray tone. The lamina cribrosa and the dark-gray patches between its bundles, corresponding to the medullated fibres, become clearly visible. In reality the visibility of the lamina cribrosa enables us to diagnose that the nerve fibres which lie before it are in part atrophic. And the ophthalmoscopic excavation which then develops does not depend on a pushing backward of the lamina cribrosa (this becomes on the contrary the more resistant), but on the fact that the lamina cribrosa is more easily seen because of the absence of its overlying nerve fibres. I have previously shown in another place that no conclusion as to the actual depth of

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[1] Leber, *l.c.*, p. 871.
[2] Albutt, cit. by Leber.
[3] Michel, *l.c.*, p. 626.

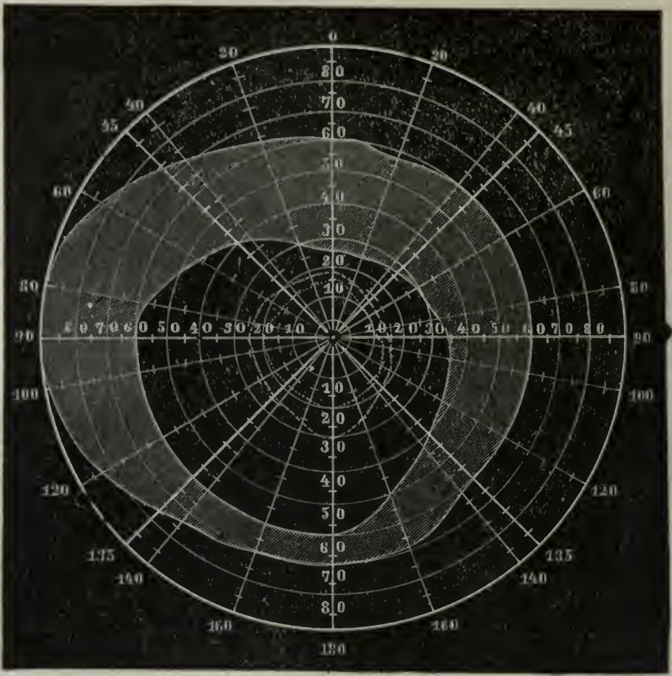

FIG. 19.—Field of vision of left eye. B., Aug. 12, 1887.

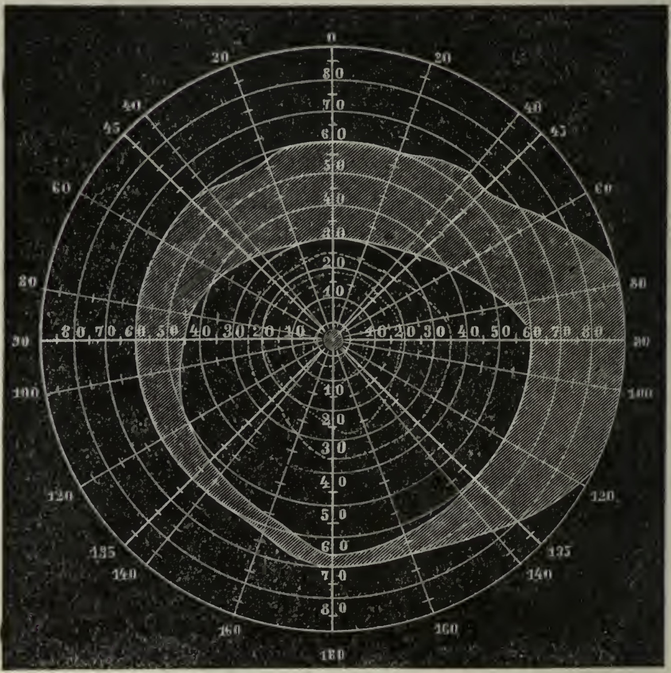

FIG. 20.—Field of vision of right eye. B.

the excavation can be drawn from the ophthalmoscopic depth.[1] At this stage the excavation appears to me of a bluish-gray color.

The more the connective tissue of the lamina cribrosa thickens, the more must the medullary substance (the nerve fibres retain their medullary sheaths up to the lamina cribrosa, or into it) diminish or become concealed by the thickened lamina cribrosa. If the nerve appear uniformly white (nacrée, Charcot) it shows an advanced atrophy.

With regard to the blood-vessels, the capillaries must be dilated, at least at the beginning of the atrophy. The central vessels undergo the following changes: (1) Arteries and veins normal; (2) arteries narrow, veins normal; (3) arteries narrow, veins dilated; (4) arteries and veins narrow. We shall speak later of the vascular affection causing these changes.

Schmeichler[2] explains both the narrowing of the arteries and the dilatation of the veins as a purely mechanical effect of the shrinking of the lamina cribrosa. Since the atrophic process, according to Leber, proceeds from the periphery of the optic nerve to its axis, one must then suppose that the vessel walls would show a change of calibre only in an advanced atrophy, which is not the case. I can therefore explain the changes in the calibre of the arteries when the atrophy is little advanced, only as a result of disease of their walls.

Changes can often be found in the region of the papilla itself. I would refer especially to the broadened pigment ring, as well as to the manifestations of choroidal changes (visibility of the vessels) about the papilla, though this latter is rare. We should not be surprised, since the anterior bundles of the lamina cribrosa pass over into the choroid, to find that the process may spread from the lamina cribrosa to the neighboring choroid. In one case a choroiditis pigmentosa existed in both eyes. This case, strictly speaking, would not come into the category of optic atrophy in tabes.

Cause of the Visual Disturbance.—Before we take up the subject of the cause of the visual disturbance in tabes, we must notice that apart from purely dioptric errors there are

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[1] Berger, "Beiträge zur Anat. d. Auges in norm. und path. Zustande," Wiesbaden, 1887, p. 151.

[2] Schmeichler, *l.c.*, p. 46.

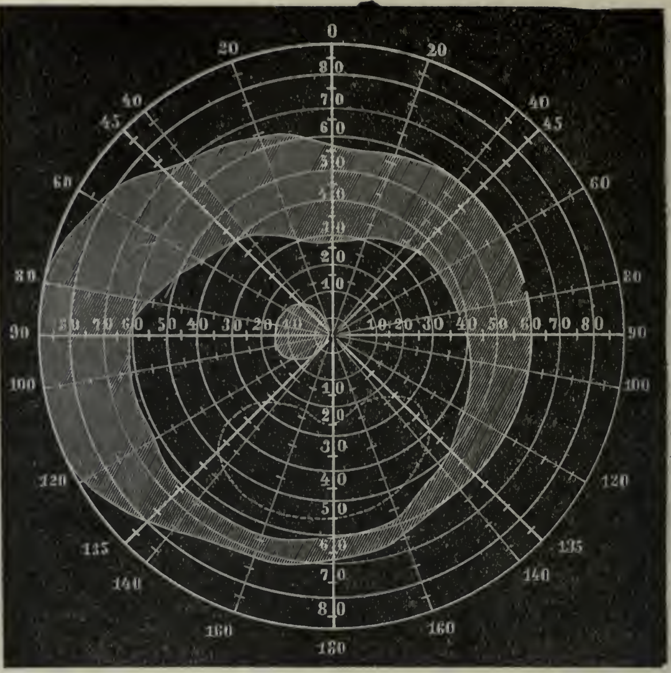

FIG. 21.—Field of vision of left eye. B., Dec. 20, 1887.

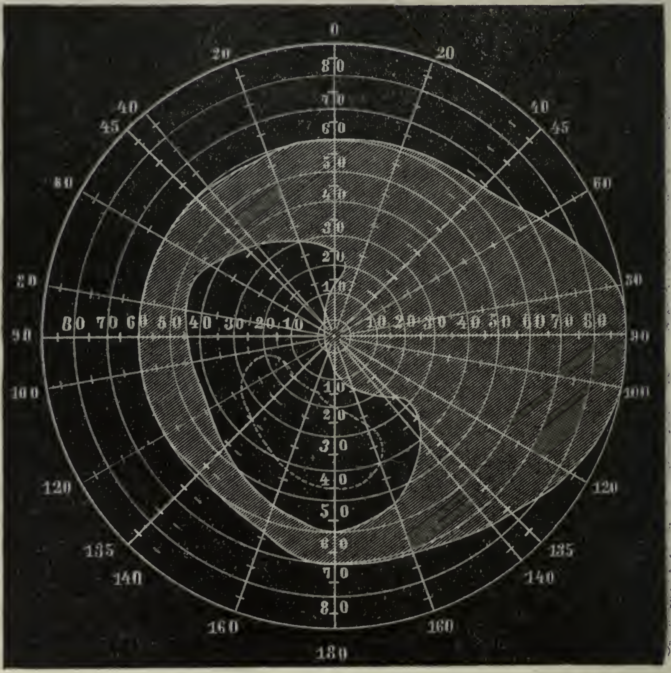

FIG. 22.—Field of vision of right eye. B.

two chief groups of functional disturbances in the optic nerve and the nerve-fibre layer: (1) those in which the fault lies in the nerve-fibre conduction; (2) those in which the functional disturbance is caused by a disturbance of nutrition.

Since the investigations of Samelsohn, Vossius, and Bunge, we are able to make a schema of the distribution of the nerve fibres in the retina. We know that the fibres of the temporal side of the nerve supply the macula and the portion of retina between it and the papilla. The fibres of the nerve immediately surrounding the central vessels supply the periphery of the retina, the fibres just outside these supply the æquatorial portion of the retina, and the fibres in the periphery of the nerve terminate in the posterior portion of the retina. Did this distribution not exist, there would be a crossing of the fibres in the papilla or in the retina noticeable on section. Since these crossings have not been found, the superposition of the fibres supplying different parts of the retina can be understood only in this manner.

Again, since no crossing of the fibres is found in the plane of the retina, we must conclude that particular sectors of the nerve send fibres to particular sectors of the retina.

Let us see now whether the defects found in the field of vision can be explained by an interruption of the fibre-conduction.

Suppose, for example, the frequent case of a peripheral contraction with sector-formed defects. The peripheral defect would correspond to a functional disturbance of the fibres in the axis of the nerve (*a*); the sector-formed defect, to a sector-formed segment (*s*) of the nerve (Fig. 23.)

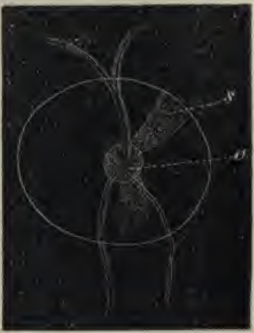

FIG. 23.

Every one will acknowledge that neither the pathological examination nor the examination with the ophthalmoscope would show such changes in the nerve. According to Leber, the atrophy of the nerve extends from the periphery toward the axis, therefore one should suppose that the posterior portion of the retina would be the first to suffer. The characteristic field in tabes would then show an enlarged blind spot which would include the macula. We have found but one such case in our investigations, and such cases are so rare that a number of authors deny their existence in tabes altogether.

The supposition that the visual disturbance in tabes is the result of a central lesion is disproved both by the form of the defect in the visual field and by the anatomical proof that the atrophy is not a descending one, but begins in the retrobulbar portion of the nerve.

We can seek the causes of the visual disturbance then, only in the vessels themselves. In reality many facts point toward vasomotor disturbances from central causes. Very probably particular vascular regions in the retina are narrowed earlier than others. I have shown in another place[1] that a slight contraction of the vessels is sufficient to cause the retina to become suddenly incapable of function. We are, however, not yet able to recognize with the ophthalmoscope narrowing of the blood columns in the periphery of the retina.

The frequent occurrence of *symmetrical* fields of vision in tabes, as well as the fact that the defect in the field of the second affected eye becomes much later symmetrical with that of the first eye, indicate vasomotor or rather vascular disturbances of central origin. The condition of the limits of the color-fields in the sector-formed defects corresponds exactly with that after partial embolism of the retinal vessels, and may therefore be considered in the sense of a vascular disturbance.

The further investigation of the question, whether a defect in the visual field corresponds to a functional disturbance of the fibre-conduction or of the vessels, would be of special importance for the theory of color-blindness.

If the Young-Helmholtz theory be correct, one must conclude that the color-blind zones conform to the regions of distribution of the nerve fibres; if the Hering theory be correct, one must conclude that the absence of the visual substance serving color-perception is confined to certain (secretory) vascular regions.

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[1] Berger and Tyrman, "Krankheiten d. Keilbeinhöhle und d. Siebbeinlabyrinthes," Wiesbaden, 1886, p. 90.

It is hard to explain by vascular causes why that particular part of the retina between the macula and the papilla retains its function longest. Many suppose that the fibres supplying this part of the retina are more resistant than the others. The cause of the disappearance of the nerve fibres lies in their compression by the shrinking lamina cribrosa. It may be that these fibres are preserved so long because they lie in the temporal side of the optic nerve where the network of the lamina cribrosa is looser.

Treatment of the Atrophy.—Since, as was shown before, various gradations are found in the course of the atrophy, and the course may be protracted and at times stationary even without treatment, we must discredit the reports of those authors who claim to arrest it by the use of drugs. Every ophthalmologist and neurologist who had had a large material had observed cases where the atrophy remained stationary for years (Hirschberg [1], Gowers, *et al.*). Galezowski believes that there is a tendency for the atrophy to become stationary in those cases where a perivasculitis exists. Gowers says that the galloping cases present a gray-gelatinous or loosened appearance of the papilla, while in slow cases the papilla is clear and excavated.

The mercurial treatment of optic atrophy has proved worthless, and even harmful (Leber : " The vision diminishes rapidly under its use "). Charcot has never seen the slightest beneficial effect from inunctions in beginning optic atrophy or in tabes in general. Under these circumstances it is to be regretted that Michel [2] recommends in cases where there has been syphilis, a methodic antisyphilitic treatment.

I have seen only slight improvement under electrical treatment—galvanization of the optic nerve and sympathetic—or under the use of iodide of potassium or arsenic. I have seen the subjective symptoms improved in one case by a strong diaphoretic treatment (Schwitzcur), together with salicylate of soda, and the patient seemed also to distinguish colors better than before. There is a case mentioned in the literature by Goldzieher, where improvement resulted from pilocarpine injections. I have had no experience with the cyanide of gold, with which Galezowski[1] had "des resultats très encourageants."

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[1] Hirschberg, *Klinische Beobachtungen aus der Augenheilanstalt*, pp. 68–71.

[2] Michel, *l.c.*, p. 629.

The strychnine treatment has been in great measure given up. It should be employed with great care, since if large doses be given the spinal symptoms may increase (lancinating pains come on, etc.). I commence the strychnine injections with $\frac{1}{2}$ *mgrm*, and increase the amount slowly. If I see that strychnine injections are not well borne, I prescribe rest in a darkened room and iodide of potassium. In some cases arsenic with iron seemed beneficial; in others it proved worthless. Nitrite of amyl, which is supposed to dilate the retinal vessels, is also of no value.

The following vision curve (Fig. 24), which represents the course of the atrophy in one patient, throws light on the results of the various treatments. I report this case in order to encourage the recording of similar vision curves, which are very instructive for the student as well as for the physician.

The patient began treatment at the end of October, 1883. Right, $V = \frac{20}{70}$; left, $V = \frac{20}{40}$; inhalations of nitrite of amyl, and nitrate of silver internally, prescribed. Dec. 20th—Right, $V = \frac{20}{70}$; left, V raised to $\frac{20}{30}$. Following this the vision grew worse. March 18th—Right, $V = \frac{20}{200}$; left, $V = \frac{20}{70}$. Strychnine treatment improved the sight of both eyes. About the middle of May—Right, $V = \frac{20}{40}$; left, $V = \frac{20}{30}$ (v. t. s.). Again the vision grew worse. At the end of November—Right, $V = \frac{10}{200}$; left, $V = \frac{20}{100}$. A diaphoretic treatment with salicylate of soda was tried for fourteen days. Dec. 10, 1884—Right, $V = \frac{10}{200}$ (yellow and blue recognized); left, $V = \frac{20}{70}$ (the same colors). Strychnine was again used, without success. April 27, 1885—Patient was color-blind in both eyes; $V = \frac{10}{200}$. Oct. 11th—V = fingers at five feet.

The Ocular Muscles.

Nystagmus, Ataxy of the Ocular Muscles.—When a tabic patient is told to move the eyes, after the movement, especially when this has been quickly performed, a slight oscillatory twitching occurs. This is most frequent in a horizontal direction.

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[1] Galezowski, Soc. de. Biologie, Apr. 7, 1883.

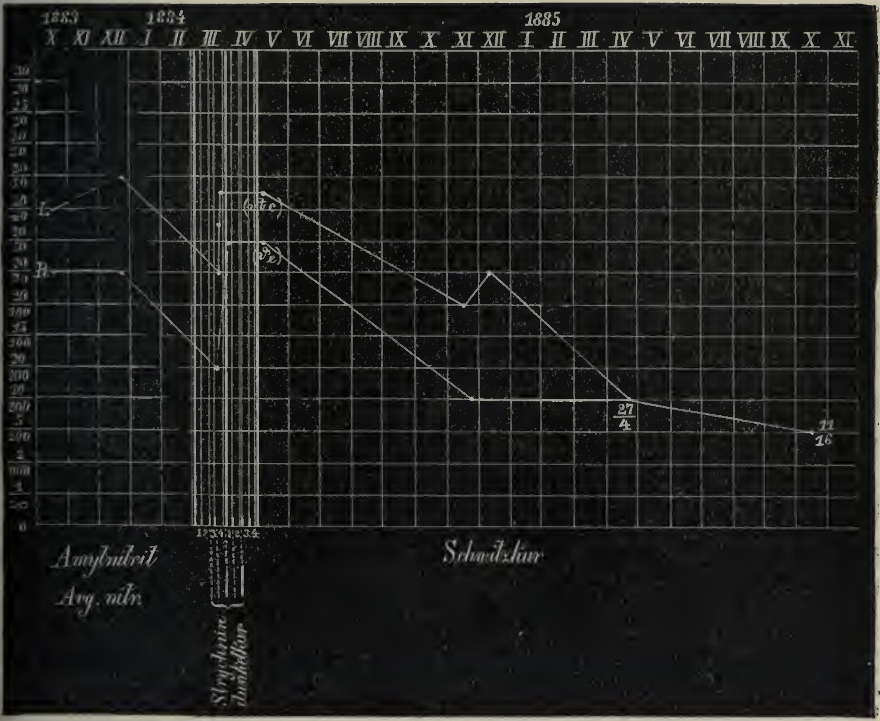

FIG. 24.—Vision curve in a case of atrophy of the optic nerve.

Friedreich, who first observed and described this symptom, named it nystagmus, and thought it an ataxy of the muscles. But since the ophthalmologists call nystagmus those clonic spasms of the ocular muscles which appear even when the patient looks straight forward, it is clear why neurologists report the frequent occurrence of nystagmus in tabes, while the ophthalmologists do not mention it (Michel), or say that it is supposed to occur (Mauthner). This nystagmus, as is known, is found also with hereditary ataxy, where Rūtimeyer[1] saw it in nine cases.

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[1] Rütimeyer, *Virchow's Archiv*, vol. xci., p. 106.

Erb[1] says again: "In the typical form of tabes these disturbances seem seldom or never to occur." According to Erb, the nystagmus belongs to the late symptoms, and appears only after the disease has lasted for years.

This trembling of the eyes after movement is very frequent in tabes. It was wanting but 6 times in 109 cases; 4 of these were in the stad. præatact., 1 in the stad. atact., and 1 in the stad. paralyt.

Much more rarely does one find in tabes a continuous nystagmus without preceding movement of the eyes. Freyer[2], in one case of tabes, found this to be the only ocular symptom.

I found this nystagmus once unilateral, when all the ocular muscles of the other eye were paralyzed, and six times bilateral.

The nystagmus was seen twice in the stad. præatact., four times in the stad. atact., and once in the stad. paralyt.

Only in two cases was there still good vision; in one case there was rotatory nystagmus; in the others horizontal.

As to the explanation of the nystagmus, when amblyopia has existed a long time, it may be explained by this alone. In those cases where the vision was normal, the nystagmus did not indicate any special cerebral localization. It is found also in quite healthy individuals.

I would remark here that I have produced nystagmus experimentally in animals by injuring the sinus rhomboidalis.

A true ataxy of the ocular muscles shows itself in tabes by the appearance of associated double images. Such cases have been described by Charcot. Althaus[3] also observed an analogous case: "The patient was scarcely able to coordinate the action of the ocular muscles." The ataxy of the ocular muscles is, however, rare. I have not seen a single case.

Paralyses of Ocular Muscles.—I have already spoken of the frequency of ocular paralysis among the patients that I have examined. It was present in 38% of my patients Erb found it in 27%, Moeli in 39.6%.

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[1] Erb, *l.c.*, p. 167.
[2] Freyer, *Berliner klin. Wochenschr.*, 1887, No 6.
[3] Althaus, *l.c.*, p. 231.

As to the stage of tabes in which the paralysis developed, Gowers found that four fifths were in the initial stage; he emphasized the tendency of lues to cause affections of the ocular muscles.

In my cases the far greater number of the ocular paralyses were in the stat. præatact. My figures would not show that luic patients are especially disposed to ocular paralysis. *The luic patients show, however, a greater tendency to permanent paralyses than the non-luic.*

I have computed the average duration of the transitory paralyses and found it:

In the non-syphilitic	2 months.
" " syphilitic	6½ "

The duration of the transitory paralyses varied between several hours and two years.

The paralysis of exterior muscles affected one eye only in twenty-four cases; in the other cases (44%) it was bilateral. The unilateral paralysis occurred ten times in syphilitic, fourteen times in non-syphilitic patients.

In twenty-eight eyes only one muscle was paralyzed, *the syphilitic being more disposed to multiple paralyses in tabes than the non-syphilitic patients.*

With regard to only one ocular muscle becoming affected in tabes, I find in the literature the following:

Erb[1] says that the oculomotor is most frequently attacked; Charcot says the same; Woinow, on the contrary, found the abducens most frequently paralyzed, and generally the left abducens.

I find that of the nerves the oculomotor is the most frequently affected, but of the single muscles, the abducens.

Of the muscles supplied by the oculomotor nerve, I found the levator palpebræ sup. most frequently affected (not including paralysis of the sympathetic, in fourteen eyes).

Paralysis of the internal rectus was found in only thirteen eyes. In this respect I stand opposed to a number of authors, *e.g.*, De Watteville,[2] who especially in the beginning of tabes found the convergent movements of the eyes frequently paralyzed, and Landolt as well. Von Graefe even had noticed the lessened tendency to fusion in tabic patients, and had referred it to a central cause.

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[1] Erb, *l.c.*, p. 179.

[2] De Watteville, Ueber die Lähmung der Convergenzbewegung der Augen im Beginne der Tabes, *Neurolog. Centralbl.*, 1887, No. 10.

The superior rectus was found paralyzed in nine eyes, and the inferior oblique in nine. Paralysis of the superior oblique was most rarely observed, being found in eight eyes (two of which eyes showed total paralysis), *more frequently on the right side.*

Total paralysis of the oculomotor nerve was found in only one case, in a non-syphilitic patient. The paralysis had affected, on the right side, the whole oculomotor nerve; on the left, all the ocular muscles, with exception of the abducens. In the other cases the paralysis of the oculomotor nerve was incomplete; in one case it was completely paralyzed, with exception of the branch supplying the levator palpebræ sup.

A large number of the paralyses of ocular muscles in tabes may be explained by the schema of Kahler and Pick for nuclear paralysis. The examinations of Déjerine have demonstrated that not all the ocular paralyses in tabes are of nuclear origin. But since the investigations of Pierret we may assume that they begin as nuclear, extend toward the periphery as a descending neuritis, and here continue after the nuclear affection is recovered from. The fact that single nerve nuclei are shown by the symptoms not to be affected, while the nerves springing from nuclei surrounding these are paralyzed, does not disprove the nuclear origin. The chronic ependymitis of the floor of the fourth ventricle is doubtless a diffuse process, but the destruction of nerve tissue in consequence of it need not extend equally deep in all parts.

Symptoms on the Part of Other Cerebral Nerves.

I was unable to examine the sense of smell in my patients; others have observed loss of the sense of smell, and also hyperosmia (Erb).

Much attention has lately been paid to paralytic symptoms in the region supplied by the fifth nerve. Charcot mentions the insensibility of the throat; Galezowski, anæsthesia of the skin about the orbit.

I found in one case, the patient being syphilitic, anæsthesia of all the twigs of the fifth nerve on the left side, as the first symptom of tabes. $V = \frac{1}{2}$. The fundus was normal, the field of vision and the color-fields greatly contracted.

Althaus described a case in which the atrophy of the optic nerve came on with a unilateral anæsthesia of the fifth nerve.

In another of my cases, a syphilitic patient in the stad. præatact., the third branch of the fifth nerve was alone paralyzed.

More frequent than anæsthesia is hyperæsthesia of the regions supplied by the fifth nerve, and cephalalgia which may be distinctly located, as, for example, in one case where the supraorbital and infraorbital nerves were decidedly sensitive to pressure. It the stad. præatact. there are often pains in the occipital region. In one case, still in the stad. præatact., there was a neuralgia of the third branch of the fifth; in another case, in the stad. atact., of the third branch.

Pierret refers the symptoms on the part of the fifth to a sclerosis in the region of the lower nucleus of the fifth in the medulla.

No neurologist denies longer that paralysis in the regions supplied by the facial nerve may occur in tabes; the opinions, however, are divided as to whether these accompanying symptoms are accidental or whether they stand in a causative relation to the tabes.

I have observed altogether six cases of facial paralysis in tabes, four of which were in non-syphilitic patients, two in syphilitic. With one exception, where the whole facial nerve was involved, the paralysis affected only the lower twigs of the nerve. The right side of the face was paralyzed five times, and the left once, one case being in the stad. præatact., one in the stad. atact., and four in the stad. paralyt.

The cases where there was difficulty in closing the lids, indicating probably an affection of the upper nucleus of the facial nerve, have already been considered.

It is difficult to understand why one should assume the paralysis of the facial nerve to be a symptom accidentally accompanying the tabes, since we know from the investigations of Kahler that the nucleus of the facial nerve may be found diseased in tabes.

Symptoms of irritation of the facial nerve have been observed, such as a continuous blinking and the making of grimaces.

I have observed, in two cases only, an affection of the hearing that could not be explained by a disturbance in the conducting apparatus.

We know to-day that the vestibular nerve is more frequently attacked in tabes than the cochlear. That tabes may be complicated by disturbances of hearing is shown by the investigations of Walton and Marie,[1] who found deafness seventeen times in eighty tabic patients at the Salpêtrière.

I have seen in five cases affection of the vagus (difficulty in swallowing, palpitation of the heart, gastralgia); in one case there were laryngeal crises.

A paralysis of the spinal accessory nerve was found once in a woman of forty-eight. Her head was turned to the right, showing a paralysis of the left accessory nerve.

I have never found glosso-pharyngeal or hypoglossal paralyses in my patients.

In one case speech articulation was difficult, yet other symptoms were present which indicated a beginning progressive paralysis.

The disturbances of speech in tabes have been studied by Friedreich. In one case he found a marked nasal tone.

Diabetes mellitus was found in two cases, both men, one in the stad. præatact., the other in the stad. atact. The urine of both patients contained 3 % of sugar.

Of important trophic disturbances I shall only mention the occurrence of joint affections in two cases; in one case there was luxation and fracture of the humerus.

Résumé.

We have seen that many of the symptoms of tabes may be explained by a lesion of the nerve nuclei in the fossa rhomboidalis and its continuation into the aquæduct of Sylvius.

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[1] Walton and Marie, *Rev. de méd.*, Jan., 1883.

The difficult closure of the lids is probably to be explained by a slight functional disturbance of the upper nucleus of the facial nerve, which is in relation with the nucleus of the fifth nerve. The myosis is paralytic, caused by paralysis of the vaso-constrictors in the medulla.

The reflex iridoplegia is produced by a lesion of the fibres running in the wall of the third ventricle to the nucleus of the sphincter pupillæ.

The diminished intraocular tension, which is found in one third of the cases, is probably the result of the lowering of the tone of the vessels. The latter is found after wounds of the medulla. The lachrymation in tabic patients is a vasomotor neurosis. It is analogous to the unilateral hyperidrosis occurring in tabes.

The sympathetic nerve, the section of which in animals causes slight ptosis and lessened ocular tension, is not the cause but the medium by which certain ocular symptoms of tabes are carried from the central nervous system to the eye.

We have seen further that in tabes a number of nerves whose nuclei lie in the medulla, may become affected. The whole facial nerve may be paralyzed, or, what is more frequent, only its lower twigs; the auditory nerve, the vagus, the fifth, and rarely the spinal accessory nerve, may suffer functionally, and, according to some authors, the glosso-pharyngeal and hypoglossal nerves as well.

It may be said in general that the nerve nuclei in the medulla nearest the middle line suffer more frequently than those further outward; further, that the nuclei in the upper portion of the fossa rhomboidalis are more frequently affected than those lower down.

An affection of the medulla is indicated by the development of diabetes mellitus in tabes, which Claude Bernard produced experimentally by cutting through the medulla; further, by the development of diabetes insipidus.

In this manner perhaps may be explained the febrile symptoms which, according to a number of observers, occur in tabes (M. Rosenthal and others). For the investigations of Tschischichin[1] have shown that the medulla is concerned in the regulation of warmth.

We have already discussed the question whether the ocular-muscle paralyses are of nuclear origin.

To the statement that, in a number of autopsies of tabic patients, no disease of the medulla has been found, we may make the objection that little attention has been given to the condition of the medulla in these cases.

On the other hand, as has been previously stated, the inflammatory process in the nerve nuclei, caused by the chronic ependymitis, may have run its course, while the process in the peripheral nerves caused by a neuritis migrans may still continue. Remembering the anatomical investigations of Pierret and Gowers (1879), we must accept this as the most probable hypothesis.

It is therefore unnecessary, as Michel[2] does, to assume the existence of hemorrhages in the central fibres and in the nuclei in order to explain the transitory paralyses of the ocular muscles which come on in the beginning of tabes.

Such hemorrhages have been found but once at autopsies. They may lie at the bottom of those very transitory paralyses of the ocular muscles which last but a few hours or days.

We know further that the antero-lateral tract, which connects the degeneration zones of the medulla with those of the cord, contains *vasomotor* fibres. Adamkiewicz has also proved that the lesions of the cord, especially in tabes, correspond in extent to the vascular zones.

Magnan found, in 1874, at the autopsy of a tabic patient, that the changes were located along the vessels. Ordonnez[3] found also that the vessels were the starting-points of the degeneration in the cord.

We have seen that from the functional disturbances of the optic nerve it may be concluded that vascular changes alone cause the disturbances.

There can be no doubt that the changes in the central vessels are not caused through the medium of the sympathetic nerve, since the section of this in animals has no influence on the width of the retinal vessels. The carotid plexus is probably the course taken by the nerves supplying the vessels of the optic nerve.

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[1] Tschischichin, quoted by Botkin, *l.c.*, p. 41.
[2] Michel, *l.c.*, p. 105.
[3] Ordonnez, quoted by Althaus, *l.c.*, p. 44.

The assumption is then admissible that the functional disturbances of the vascular regions of the optic nerve and posterior columns of the cord are caused by a disease of the nerve-centres in the fossa rhomboidalis, resulting from an ependymitis of the medulla.

Why particular vascular centres are specially disposed to a simultaneous affection, may perhaps be explained by their more exposed position rather than by their peculiar nutritive relations.

We now have the following points to consider:

(1) Whether we are justified in assuming the vascular centres to be located in the medulla.

(2) That in consequence of disease of the nerve centres vascular changes can develop.

(3) That vascular changes occur in the various nerve regions which are the seat of the morbid process in tabes.

(4) That the vascular changes are also the cause of the disease of the nerve tissue.

(5) Whether trophic nerves cannot cause simultaneously the changes in the affected nerve regions and in the vessels.

1. Do the Vascular Centres Lie in the Medulla?

We know that the action of the blood-vessels is to be explained by the function of the vaso-dilators and the vaso-constrictors. Concerning the latter, Cohnheim says: "It can no longer be denied that there exist in different portions of the cord, centres which control the vascular nerves that come off below; the constrictors have their chief centre in the medulla—a centre which is in a continuous state of irritation. We know that this vasomotor centre is strongly stimulated by dyspnœic blood; anæmia has the same effect, whether it be general or localized in the medulla."

The medulla has also an influence on the vascular tone. Brown-Séquard has shown that the medulla has a regulating influence on the filling of its own vessels and those of the cord.

2. Do Vascular Changes Develop in Consequence of Disease of the Nerve Centres?

Our knowledge in respect to this point is still very limited.

Eppinger,[1] in Klebs' laboratory, studied a disease of the vessels similar to atheromatous degeneration, which is dependent on central changes; "in the paralytic insane, where so many striking vascular manifestations occur in the brain, it may not be too fanciful to suppose that the vessels stimulated by the vasomotors become diseased in consequence of disease of the unknown innervation centres, and that their coats undergo a change."

An experiment of Adamkiewicz[2] also indicates the dependence of vascular changes on central alterations. When one cerebral hemisphere was compressed, the eye of the opposite side showed diminished tension, hyperæmia of the conjunctiva and uveal tract, and later a plastic exudation into the latter.

3. The Disease of the Vessels in the Nerve Regions which are Affected in Tabes.

Kahler found a number of wide vessels with thickened walls in the gray substance about the aquæduct of Sylvius.

In the cord "the vessels were found for the most part thickened, with walls rich in nuclei (Erb[3]). Rumpf, Strümpell, and Kraus described the disease of the vessels in the cord as a thickening of the coats external to the endothelium.

Buzzard[4] found the vessels increased in number and widened, with thickened walls and often gaping lumen, apparently the centre of the sclerotic changes.

Bevan Lewis found a periarteritis and ampullaform dilatation of the vessels, and again contractions.

Vascular changes were found by Oppenheim in peripheral neuritis. He found the vessels for the most part obliterated, their walls thickened and sclerotic.

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[1] Eppinger, *Prager Vierteljahrsschr. f. prakt. Heilk.*, 1875, p. 67.

[2] Adamkiewicz, *Wiener Sitzungsberichte*, vol. iii., 1884, p. 231.

[3] Erb, *l.c.*, p. 139.

[4] Buzzard, *Brain*, 1884.

These vascular changes have been little studied. Leber[1] found the vessels "but slightly changed," the larger vessels normal.

That the vessels are concerned in the extension of the disease is shown by the following statement of Leber: "Here and there in the neighborhood of single vessels which passed from the inner sheath into the optic nerve were thick masses of small lymphoid cells."

Reymond and Arthaud found also in the sympathetic nerve thickening of the vessels.

In my own investigations[2] in secondary ascending atrophy of the optic nerve, which shows changes analogous to those found in tabes, I could consider the disease of the vessel walls only as an endarteritis obliterans.

Michel[3] refers the diseases of the cord following syphilitic affection to an endarteritis and perivasculitis.

4. The Vascular Affections are the Cause of the Degeneration of the Nerve Elements.

I have said before:[4] "The increase of connective tissue as well as the simultaneous atrophy of the nerve elements depends on the changed nutritive conditions of the optic nerve caused by the obliterating inflammation of the vessels."

The results of the narrowing of the vessels has been thoroughly studied by Cohnheim.[5] There is caused a slowing of the blood-current, "the specifically heavy blood corpuscles do not remain suspended in the blood-stream, but rest heaped up in the capillaries." This latter leads to diapedesis of the white corpuscles.

Experimental studies on the influence of anæmia on the nerve tissue of the cord have been made by Spronck. He found the final result to be a necrosis of the ganglion-cells and their processes, and a proliferation of the neuroglia cells (karyokinetic figures appeared in them).

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[1] Leber, *Graefe's Archiv*, vol. xiv., 2, p. 188.

[2] Berger, "Beiträge z. Anat. d. Auges in norm. und path. Zustande," Wiesbaden, 1887, p. 134.

[3] Michel, *l.c.*, p. 629.

[4] Berger, "Beiträge z. Anat. d. Auges in norm. und path. Zustande," p. 134.

[5] Cohnheim, *l.c.*, p. 115.

5. Are Changes of Vascular or Trophic Nerves which have the same Course as the Vessels the Causes of the Degeneration of the Nerves?

Although the existence of trophic nerves has not been demonstrated anatomically, a number of the best investigators believe them to exist. There are a number of trophic disturbances occurring with disease of the brain and cord (joint affections, decubitus, etc.) which cannot be explained as being caused by vasomotor influences. From disease of the nervous system which causes a paralysis of the vasomotors, may result a hyperæmia, but never trophic changes (Charcot).[1]

The symmetrical development of trophic changes, such as the becoming gray and falling of the hair, skin affections, etc., is supposed to indicate the existence of trophic nerves. Very probably the anterior cornua of the cord form the trophic centres for the trunk and extremities. Jarisch[2] found in fact changes in the anterior cornua in a number of skin affections.

Joseph[3] has produced circumscribed alopecia experimentally in cats by the excision of the spinal ganglia with a bit of the anterior and posterior nerve-roots.

In our case, the changes in the cord in tabes and those experimentally produced so correspond that it is not necessary to assume a direct influencing of the tissue by the trophic changes in tabes. It is otherwise with the changes in the vessels themselves. We must here decide whether these be produced by the vasomotors themselves or by their own trophic nerves, which have the same central origin as the former. This question is, however, of little importance as regards our knowledge of the relation of the various nerve regions affected in tabes.

Where do the Vessel Centres of the Optic Nerve Lie?

We know that the atrophy of the optic nerve in spinal diseases is most frequent in tabes, but is still found in the acute form of myelitis, in spastic spinal paralysis, and after injuries of the cord. In progressive bulbar paralysis *the atrophy of the optic nerve comes on at the same time with paralysis of the facial and abducens nerves.* Michel[1] says rightly: "The higher the location of the injury of the cord, the sooner develop disturbances in the eye."

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[1] Charcot, "Leçons sur les maladies du système nerveux," 1877, vol. i., p. 151.

[2] Jarisch, Beitrag zur Path. d. Hautkrankheiten, *Wiener Anzeiger*, 1881, pp. 44–46.

[3] Joseph, *Virchow's Archiv*, vol. cvii., part i.

In order to answer the question, where may the vessel centre for the optic nerve lie? I have compared the cases of paralyses of the nerves whose nuclei are in the floor of the fossa rhomboidalis or its continuations where atrophy of the optic nerve existed, with the cases of other nerve paralyses without optic atrophy. If the lesion of a particular region of the fossa rhomboidalis be the cause of the atrophy of the optic nerve, then the nerve nuclei nearest this region will be most frequently affected.

My results were, in general, that the paralyses of the ocular muscles whose nuclei lie in the posterior portion of the oculomotor nucleus are more frequent in connection with atrophy of the optic nerve than without it.

Since the level of the facial-abducens nucleus represents the lower limit for the occurrence of atrophy of the optic nerve in diseases of the medulla, the experimental investigations as to the vessel centre of the optic nerve had no great region to cover. The rarity of the affection of the fifth nerve narrows considerably the field to be investigated.

Experimental Investigations.

My experiments, in which I cut different parts of the medulla oblongata, showed that:

1. Nystagmus may develop;
2. Mydriasis as well as myosis (with oval pupil) may appear;
3. As a result of injury to the medulla, hemorrhages are found in particular vascular regions of the cord (from dilatation of the vessels), in the third experiment especially on the outer surface of the posterior columns, in the fourth experiment, which produced an effect complementary to

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[1] Michel, *l.c.*, p. 628.

that of the third, on the outer surface of the pia mater of the anterior and lateral tracts;

4. After injury to the medulla changes in the retinal vessels may come on.

I am far from regarding these investigations as a direct proof of my explanation of the symptom-complex of tabes; I believe, however, that they support it. I leave to other investigators the problem of the influence of the medulla on the vessels of the cord and retina.

ON THE NUMBERING OF PRISMATIC GLASSES USED IN OPHTHALMOLOGY.

BY DR. EDM. LANDOLT.

Translated by Dr. PAIGE.

IN the year 1887 Dr. E. Jackson[1] proposed to the oculists at the International Medical Congress a more rational plan for numbering prisms used in ophthalmology. I seconded the proposition as a very correct and timely one, and had the honor of being chosen, with Drs. Jackson and Burnett, as a member of a committee whose duty it should be to submit such a plan at the next International Congress. I did this at Heidelberg in the year 1888.[2] It was not, however, extensively discussed, on account of lack of time. So I introduced it once more at the International Medical Congress at Berlin (1890). The exhaustive treatment and proper solution of this question, however, seem of so great importance that I would like to briefly discuss it in these ARCHIVES.

Fifteen years ago we changed the numbering of spectacle glasses, then based upon their **form**, to a more rational system based upon their **force**.

Practice has proved this to be a step in the right direction. It is but logical to give the prisms the benefit of the same advantage, and in place of designating them by their refractive angle to designate them according to their *refractive power*.

We use prisms to change the direction of rays of light so as to displace retinal images, or the apparent position of objects looked at. They carry numbers which have only an indirect relation to this function, and consequently do not inform us as to the service we require of them. Let us replace this numbering, which is of no practical use, by a sign which will tell us how many degrees a luminous ray in passing through a prism is deflected from its original direction. In a word, in place of designating the prism by its angle of refraction, let us designate it by the angle of deviation which it produces. We shall know then that a strabismus of 15° requires a prism of 15° to correct it; that, when a diplopia is corrected by a prism of 10°, it corresponds to a strabismus of 10°, and so on.

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[1] Report of the Congress at Washington, 1887, p. 785.

[2] Report of the Congress at Heidelberg, 1888, p. 437.

It is true that the same prism deflects luminous rays a little differently, according to the angle under which they fall upon it. From this point of view two positions of the prism must be taken into consideration: the one in which the luminous ray is perpendicular to one of the surfaces of the prism, and the other in which it traverses the prism perpendicular to the bisecting plane of the refractive angle. In the latter case the prism produces its *minimum of deviation*, while in the former the deviation is somewhat greater.

Thus, with an index of refraction of 1.53 and a refractive angle of 5°, the minimum of deviation is 2° 40′ 6″, and the deviation of a ray which strikes the prism at a right angle is 2° 40′ 40″; the difference is 34 seconds.

For a prism of a refractive angle 21° 50′ aperture and of the same index of refraction, the difference will be 1°. The difference is very slight and may be neglected for most practical purposes. Nevertheless, for correctness' sake, we may decide which of the two positions we should choose to determine the angle of deviation. This angle will be the number of the prism.

Greater simplicity has been mentioned in favor of the first position. There is, indeed, deflection of light only at one of the surfaces of the prism; the position of the minimum of deviation is, however, that in which the prisms are generally used and in which their secondary effects are least noticeable. It therefore seems more desirable to choose the angle of the minimum of deviation for the designation of prisms.

Since Dr. Jackson's proposition two other plans have been brought forward, both very ingenious, but to my mind much less practical.

The first is that of Dr. W. S. Dennett.[1] The author wishes to take for the unit of prismatic refraction the hundredth part of the radian, giving it the name of "Centrad." This angle of deviation would be 34° 22′ for flint glass, with an index of refraction of 1.57. In this case the number of the prism would be nearly that of its refractive angle, and the prisms in our trial cases might, according to this author, serve just as they are, their numbers expressing directly "Centrads." But the glass which we use in practice has never 1.57 but at the utmost 1.54 for its index of refraction.

On the other hand, all the promoters of reform in the numbering of spectacle glasses, as well as of prismatic glasses, have not failed to advance as one of their most powerful arguments, the uncertainty of the index of refraction of our glasses, so that, having the same shape and consequently the same number, the glasses differ notably in refractive power.[2] The possibility of sometimes making use of the existing numbers of prisms is therefore rather a defect than an advantage.

The other proposition comes from Mr. Prentice.[3] This author would take for his measure of the prism the tangent of the deviation which it produces for a radius of one metre. According to him, the unit (the No. 1) would be a prism which, at this distance, produces an apparent displacement of 1 *cm*. He calls it a "prism-dioptry." The No. 2 would correspond to a prism which would produce a deflection of 2 *cm* (two "prism-dioptries"), and so on.

The advantage of this system, as well as that of Dennett would be its relation with the metre-angle. Thus, the half of the distance which separates the centres of rotation of the two eyes, the half of the *base line*, is the *tangent of the metre-angle*, the ray being = 1 metre. Each centimetre of this value corresponds to one "prism-dioptry." Take for example a base line = 6 *cm:* half = 3 *cm;* the metre angle will be equal to 3 "prism-dioptries" of (No. 3) Prentice's system.

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[1] William S. Dennett, M.D.: A New Method of Numbering Prisms.—Trans. Amer. Ophth. Soc., 1889.

[2] See Prentice, *l.c.*, p. 69.

[3] Charles F. Prentice: A Metric System of Numbering and Measuring Prisms.—ARCH. OF OPHTH., xix., NO. 1, 1890.

Such are the views of these authors and also of my colleagues of the Washington Congress Committee, one of whom, Dr. Jackson, has been converted to Dennett's plan, while Dr. Burnett[1] gives the preference to that of Mr. Prentice.

I must confess I cannot agree with either of them; although I acknowledge the value of the two systems. I find them inferior to the numbering by the simple angle of deviation.

The advantage of the connection between the two systems and the metre-angle does not appear to me very great. No one is more favorable than I to the excellent principles of Professor Nagel, which every day renders us great assistance in practice, but the metre-angle was invented to measure the power rather than the angle of convergence, and it is better, if need be, to convert metre-angles into degrees, than to reduce angles which have an absolute value to metre-angles, these being variable in different individuals. We must also remember that disturbances of mobility are not confined to changes of convergence, and that strabismus does not occur in the horizontal plane only. The metre-angle clearly, therefore, is not suitable for the determination of deviations of the eyes in height.

The two systems proposed have furthermore the great drawback of requiring a long explanation, and that because they are artificial, and are not based upon values in everyday use. We all have learned to calculate in degrees, but who among us is familiar with the "radian"? The tangent, called "prism-dioptry," also requires explanation, and can only be comprehended by its reduction to degrees, with which we are acquainted.

In any case, it appears inadmissible to us to choose for the designation of prisms any other measure than that which serves to estimate the anomalies of mobility which they are called upon to measure and correct. Now, we appreciate and express the excursions of the eyes as well as their anomalies in angular degrees; the field of fixation, the angle of strabismus, are objectively measured by instruments which (like the perimeters, ophthalmometers, strabometers, etc.) are divided into degrees. The results of these measurements must correspond with those of the subjective measurements made with prismatic glasses.

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[1] Swan M. Burnett: Introductory Remarks.—*L. c.* of Mr. Prentice.

To what confusion would we expose ourselves, and how incomplete would be our reform, if we were to choose in each case a different unit of measurement.

Whatever advantages the radian may possess for higher physiology and the tangent for certain special cases, for our purposes there is undoubtedly no more simple or practical designation of prisms than that in degrees of the minimum of deviation.

SYSTEMATIC REPORT ON THE PROGRESS OF OPHTHALMOLOGY DURING THE FOURTH QUARTER OF THE YEAR 1889.

By H. MAGNUS, Breslau; C. HORSTMANN, and
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WITH THE CO-OPERATION OF

S. M. Burnett, Washington; Dantone, Rome; Hirschmann, Charkow;
E. Marckwort, Antwerp; P. v. Mittelstädt, Metz; L. Werner,
Dublin; C. H. A. Westhoff, Amsterdam; Schiötz and
Ole Bull, Christiania, etc.

Sections I.–V. Reviewed by Prof. H. MAGNUS.

I.—GENERAL OPHTHALMOLOGICAL LITERATURE.

a.—STATISTICAL PAPERS.

400. **Dubrovo.** Report on the eye patients in the Government Hospital at Klodimit for 1888. *Wjestnik Ophth.*, May 4, 1889. Patients, 690; operations, 91.

401. **Klein.** Seventh annual report of Dr. Klein's Ophthalmic Hospital at Neisse, Feb. 1, 1889, to Jan. 31, 1890. Neisse, 1890. New patients, 1,282; operations, 146.

402. **Lapersonne.** Compte rendu annuel de la clinique ophtalmogique de la faculté de médecine de Lille pour l'année 1888–1889. Lille, 1889.

403. **Massachusetts Charitable Eye and Ear Infirmary.** Sixty-fourth annual report, for the year 1889.

404. **Parinaud.** Compte rendu du service ophtalmologique de la Salpêtrière. *Soc. d'opht. de Paris*, Oct. 20, 1889.

Lapersonne (402) publishes a report on the ophthalmic clinic at Lille. Number of patients, 1,245; of operations, 141. He treats granulations with sublimate (1 : 200), which he brushes upon the everted lids. Sometimes he also uses jequirity and the galvano-cautery.

The number of patients treated at the MASSACHUSETTS EYE AND EAR INFIRMARY (403) was 10,225. Dr. Shaw tabulates 147 cataract extractions in a very careful manner. Of these there were 97 by Graefe's method, 23 simple, 11 simple with excision of a circular piece of iris, 12 by suction, 3 linear, and 1 Le Brun. Among those according to Graefe, there were 3 failures, 5 had quantitative perception of light; of the simple, one saw motion of the hand; all the rest had vision from 0.1 to 0.7, BURNETT.

PARINAUD (404) discusses the eye-symptoms observed in patients with nervous diseases at the Salpêtrière. The numerous manifestations of hysteria in particular are described at length. Parinaud also saw nystagmus in hysteria. In the discussion, Meyer expresses himself rather skeptically in regard to Parinaud's observations. MARCKWORT.

II.—GENERAL PATHOLOGY, DIAGNOSIS, AND THERAPEUTICS.

a.—GENERAL PATHOLOGY.

405. HIRSCHBERG. On the formation of drops in the lens. *Centralbl. f. prakt. Augenhk.*, 1889, p. 330.

406. TROUSSEAU. Lupus et tuberculose oculaires. *Arch. d'opht.*, vol. ix., No. 6, p. 514.

HIRSCHBERG (405) remarks that the original form of lenticular opacity is the drop. In traumatic cataract also drops or globules can be shown to exist in the lens.

TROUSSEAU (406) inoculated the anterior chamber and cornea of rabbits with lupous masses taken from the conjunctiva, nose, and cheek of a woman affected with lupus, and observed in the first case typical tuberculosis of the iris 12 days after absorption. In the second, also, tubercles developed, but only a few, which disappeared after three months without a trace (except slight ectasia of the cornea at the point in question), while the tubercles of the iris continued to flourish. *Under the microscope the lupus showed all the characteristics of genuine tuberculosis, besides Koch's bacilli*, and also produced tuberculosis by inoculation, is therefore identical with this disease. The cornea is a poorer soil for tuberculosis than the iris—an important fact from a clinical point of view. The author has treated two women with corneal tuberculosis for some time, while in other cases of tuberculosis of the iris he had to enucleate at an early date. The danger of general infection is much less in tuberculosis of the cornea than that of the iris. v. MITTELSTÄDT.

b.—GENERAL DIAGNOSIS AND THERAPEUTICS.

407. GALEZOWSKI. Traitement des migraines périorbitaires et oculaires par l'application de l'électricité statique. *Rec. d'opht.*, 1889, No. 10, p. 601.

408. NIEDEN. The application of Rotter's pastilles in ophthalmology. *Centralbl. f. prakt. Augenhk.*, 1889, p. 356.

409. SIFFRE. De l'énucléation dans la panophtalmie. *Thèse de Montpellier*, 1889.

410. THOMALLA. On coloring the diseased cornea with fluorescein, and the value of this method in making a diagnosis and differential diagnosis. *Centralbl. f. prakt. Augenhk.*, 1889, pp. 332 and 353.

411. DE WECKER. L'abus de l'énucléation. *Ann. d'ocul.*, vol. cii., p. 192.

GALEZOWSKI (407) recommends static electricity in neuralgia and rheumatic affections, especially in periorbital headache.

NIEDEN (408) recommends Rotter's pastilles, which consist of zinc. sulfo-carbol., zinc. chlorat., $\overline{aa}$., 0.6 ; acid. boric., 0.4 ; acid. salicyl., 0.1 ; acid. citr., 0.1 ; thymol., 0.1. These remedies are made up into pastilles, one to be dissolved in 250 *grms.* of water. The antiseptic value of such a solution is superior to that of a 1 % solution of sublimate.

DE WECKER (411) is in favor of greatly restricting the indications for enucleation. He has preserved many eyes which ordinarily would have been enucleated by repeated sclerotomies and punctures in the equatorial region. In this manner he treats painful eyes, glaucoma, and even hemorrhagic glaucoma. Staphylomatous eyes, in which the ciliary region is also distended, are treated with repeated punctures ; enucleation, in such cases, he considers forbidden. MARCKWORT.

III.—INSTRUMENTS AND REMEDIES.

a.—INSTRUMENTS.

412. DENTZ. Jets over het gebruik van creoline in de oogheelkunde. *Nederlandsch Tijdschrift voor Geneeskunde*, vol. i., p. 31.

413. Wicherkiewicz. Sur la valeur des mydriatiques et des myotiques pour l'amélioration de la vue dans les opacités fixes du système dioptrique de l'œil. *Arch. d'opht.*, vol. ix., 6, p. 520.

Dentz (412) recommends a ¼ % solution of creoline in scrofulous ophthalmia, in blennorrhoic conjunctivitis, and trachoma. Excellent results were obtained in inflammations of the lachrymal sac by injecting a few drops of a 1 % solution, after previous dilatation of the strictures with probes. The only objection is the great pain. Westhoff.

Wicherkiewicz (413) is opposed to the use of mydriatics in the cases in question, but recommends myotics in peripheral opacitics projecting into the pupils, particularly pilocarpine in ½ to 1 % solutions. v. Mittelstädt.

IV.—ANATOMY.

a.—NERVES AND CENTRAL ORGAN.

414. Barabaschow. Contribution to the knowledge of the structure of the retina. *Inaug. Diss.*, Charkow, 1889.

The results of Barabaschow's (414) investigations may be summed up as follows: The first layer of the retina, beginning at the inner surface—the margo limitans of Schwalbe—must be designated as the "zone of the inner limiting layers," and is not a true layer. The zone of "nerve fibres" consists of fibres which in mammals possess an axis-cylinder and a thin, even sheath. One part of the fibres of this layer is composed of the inner, ramifying offsets of the ganglion opticum; the other is formed of delicate nerve fibres which come from the outer layers of the retina, pass between the cells of the third vertically into the second layer, where, taking a sudden turn, they are lost among the other fibres. The fourth inner reticulated layer consists of two kinds of fibres: some, belonging to the substratum, possess numerous offsets and form a reticulum, which, in birds, is particularly well developed. The second kind of fibres, delicate, varicose, passes through the meshes of the reticulum, and forms, by division and anastomosis, a network of nerve fibres, which receives its fibres partly from the outer offsets of the cells of the third layer, partly from the inner offsets of the cells of the fifth layer. The author did not observe any independent cell-elements in this layer.

The sixth "outer reticulated layer" consists: (1) Of the supporting-tissue; (2) nerve fibres, and (3) nerve cells. The supporting-tissue is composed of stellate cells, Krause's membrana fenestrata, which are connected with Müller's fibres. (The wavy offsets of these stellate cells make the impression of thick trabeculæ, and cause the dark lines which Dogiel considered cross-sections of the lateral offsets of his "winged cells." The nerve fibres are ramifications of the peripheral offsets of the bipolar and multipolar cells of the inner nuclear layer (outer zone of the nerve cells of the author). The cells of this layer have a large bright nucleus, and lie in the meshes of the membrana fenestrata. The author calls them "end cells," and considers them the endings of the fibres of the optic nerve. In the outer part of the body of these cells he found groups of large granules, the "granular nodules" of Dogiel. The broadened basal parts of the visual cells are in close apposition to this part of the protoplasm of the end cells. This protoplasm subdivides into many offsets. Safranine, carmine, and picrocarmine intensely stain the nuclei of the end cells, when the retina has been treated with osmic acid, but not the protoplasm. Osmic acid imparts a grayish-yellow color to the protoplasm, and leaves the nucleus bright. All this encourages the author in his belief that these formations are the terminations of the fibres of the optic nerve, in which the vibrations of light are transmuted into irritation of the nerve.

Müller's fibres send out lateral offsets even in the fibre layer. In the inner reticulated layer they form the supporting-tissue, the main trunks retaining their radiate direction. In the layer of the nuclei of the visual cells the flat offsets of the supporting fibres form nests. The nests of the outermost layer contain mainly the nuclei of the cones; the nests of the layers contain the nuclei of the rods. The nests of the nuclei of the cones lie in one plane (lamina fenestrata of the author), and have thickened edges, from which delicate sheaths with facets along their length pass over the inner parts of the visual cells. After prolonged maceration the sheaths break up into fibres (fibre-basket of M. Schulze). If the maceration is prolonged still further the nests also split up into fibres and take on the appearance of brushes. This result is therefore an artificial one. The author believes with Ranvier that the different cells exercise different functions. The hypotheses of the author are not adapted for review.

HIRSCHMANN.

V.—PHYSIOLOGY.

a.—RETINA.

415. Bjerrum. Om en tilföcelse til den sadranlige synsfelt undersögelse samt om synsfeltet ned glaukom. *Nord. ophth. Tidsskrift*, vol. ii., No. 3, 1889.

416. Exner. The retinal image in the eyes of insects. *Sitzungsberichte d. Kaiserl. Akad. d. Wissensch.*, 1889, vol. xcviii., Nos. 1–4.

Bjerrum (415) has tested the visual field with small squares of ivory varying from 20 to 1 *mm*, and finds them very useful in detecting small scotomas and contractions which otherwise would have escaped notice. In glaucoma he finds that the contraction goes to the blind spot. Schiötz.

Exner (416) shows that insects do not see through numerous inverted, but through one upright image. Exner was able to observe this upright image directly.

b.—ACCOMMODATION.

417. Gallenga. Della misura del tempo nella determinazione dell' accommodamento relativo. *Ateneo med. Parmense*, vol. iii., 2.

Gallenga (417) has found in his measurements of the relative accommodation that its movement from the relative far- to the relative near-point is more rapid than in the opposite direction, the time varying between $\frac{2}{100}$ and $\frac{8}{100}$ of a minute.

Dantone.

Sections VI.–XI. Reviewed by Prof. HORSTMANN.

VI.—ANOMALIES OF REFRACTION AND ACCOMMODATION.

418. Cotter, C. O. Correcting the whole error of refraction and the necessity for the use of mydriatics. *Amer. Jour. Ophth.*, 1889, No. 7, p. 204.

419. Coggin. Notes on some vagaries of accommodation. *Amer. Ophth. Soc.*, 1889.

420. Holt. The efficiency of hydrobromate of homatropine in controlling the accommodation for the purpose of fitting glasses. *Amer. Ophth. Soc.*, 1889.

421. Holt. Effect on the accommodation of a patient's eye by looking into a mirror set obliquely in the room during ophthalmoscopic examination. *Ibid.*

422. Jackson, E. Ametropia as determined under complete mydriasis. *Ibid.*

423. Kirchner, M. Investigations on the development of myopia. *Zeitschr. f. Hygieine*, 1889, vol. vii.

424. Schmidt-Rimpler, H. The question of school-myopia. *v. Graefe's Arch. f. Ophth.*, vol. xxxv., 4, p. 249.

425. Scimeni. Sull'astigmatismo corneale in sequito ad estrazione di cateratta. *Ann. di ottalm.*, vol. xviii., 4–5, p. 299.

426. Sym, W. G. Diphtheritic paralysis of accommodation. *Edinburgh Med. Jour.*, Nov., 1889.

427. Theobald, S. The employment of oleate of veratria to to facilitate the determination of errors of refraction. *Amer. Ophth. Soc.*, 1889.

In cases in which the total amount of hypermetropia is to be determined, Holt (420) does not consider homatropine as effective as atropine, as it does not completely paralyze the accommodation.

In determining refraction with the ophthalmoscope Holt (421) advises to let the patient look into a mirror placed to one side, as the eye is thus adjusted for a greater distance and accommodates less, while the pupil becomes wider.

Jackson (422) examined one thousand eyes before and after complete paralysis of accommodation with atropine, and found that in almost every case there had been a change in the refraction towards hypermetropia.

Kirchner (423) examined the eyes of the Friedrich and Leibniz high-schools at Berlin, and comes to the following conclusions: Heredity plays an important part in the development of myopia. The preparation for the pursuit in life to be followed has an important bearing on its development, frequent and continuous accommodation and convergence being necessary. Improperly arranged seats, poor light, and books and materials which by their color and shape make too great a demand upon the eyes, are also important factors. He advises to see to it that the school-rooms are well lighted, even the darkest part receiving an illumination not less than that of ten metre-candles. Proper seats must be provided, and all children should be examined on entering school and at stated intervals. Work at home must be reduced to the lowest limit.

Schmidt-Rimpler (424) examined the eyes of 1,710 scholars at different high-schools, and three and one-half years later 702 of them a second time. He found that the percentage of myopia increased in the higher classes. In the development of the higher degrees heredity played a part. The pigmentary changes close to the papilla or outside a closed conus increased with the degree of myopia. The number and size of the crescents also increased with the degree of myopia, although even in high degrees there were no crescents in 20 per cent. Of the 702 scholars, 25.3 per cent. showed an increase of refraction.

Among ninety-five patients operated on for cataract, SCIMENI (425) found astigmatism in thirty-nine up to 3 D with the ophthalmometer, while in fifty-six it varied between 3 and 18 D. The author proves mathematically that mere diminution of curvature of the meridian perpendicular to the section cannot cause the higher degrees of astigmatism by displacing the edge of the wound, and that increased curvature of the opposite meridian must therefore also take place. This change in both meridians was proven by twenty-six measurements, made before and after the operations. In some cases, in which there had been astigmatism of 1 D or less before the operation, the author found it increased afterwards from 3 to 10 D. He seeks the cause of the increase of curvature of the horizontal meridian in the pressure of the recti muscles upon the globe, partly relaxed by the temporary absence of the anterior chamber and the removal of the lens. This astigmatism, however, diminishes in course of time, as he also found by direct measurement. DANTONE.

SYM (426) reports on seven cases of diphtheritic paralysis of accommodation. He thinks that it is due to poisoning of the nerve centre, like in paralysis due to sausage- and fish-poisoning. WERNER.

In determining refraction, especially of astigmatic patients, THEOBALD (427) uses oleum veratrini, a 1-per-cent. solution of which he lets them rub in at the temple for three or four days. In regular astigmatism he corrects only the manifest ametropia, but in astigmatism against the rule he makes full correction.

VII.—LIDS.

428. BERGER. Deux nouvelles opérations de blepharoplastie par la méthode italienne modifiée. *Rec. d'opht.*, 1889, No. 11, p. 650.

429. GALLENGA. Fibroma sclerosante della palpebra superi-

ore sinistra con infiltrazione calcarea e placche osteomatose. *Ann. di ottalm.*, vol. xviii., 4–5, p. 372.

430. HUTCHINSON, J. Syphilitic disease of the eyelids. *Ophth. Hosp. Rep.*, vol. xii., 2.

431. MAXIMOWITSCH, M. J. Simplified operation of ectropium of the lower lid. *Wjestnik Ophth.*, 1889, No. 6, p. 510.

432. TAMAMSCHEW, J. New investigations on the pathology and therapeutics of diseases of the lids in trichiasis and distichiasis. *Wojenno Med. Jour.*, 1889, No. 9.

433. VAN DUYSE. Elephantiasis de la paupière supérieure. *Ann. d'ocul.*, cii., p. 157.

434. DE VINCENTIIS. Trapanazione del margine ciliare. *Ann. di ottalm.*, vol. xviii., 4–5, p. 379.

BERGER (428)successfully transplanted a pedunculated flap from the upper arm in two cases. In the first the eye was closed at the beginning of the operation by tarsoraphy and not opened until eleven and a half months later. In the other case only the outer two thirds were closed, the other eye being blind. MARCKWORT.

GALLENGA (429) describes a tumor of the upper lid which he removed at the Turin clinic. It was 3 *cm* long and wide, and its thickness varied between 0.8 and 1.4 *cm.* It proved to consist mainly of connective-tissue fibres, with some deposits of lime, and small bony plates at the posterior wall. DANTONE.

The syphilitic affections of the eyelids occur, according to HUTCHINSON (430), primarily in the form of the lid-chancre. It generally appears as the genuine ulcus durum, and the accompanying bubo usually causes no pain. Later tertiary and secondary affections are most frequently found in persons who have suffered with severe syphilis, eruptions of the skin, and ulcerations of the gums. The upper lid is generally the one affected, though both may be attacked. The gummous ulcers show great powers of resistance against antisyphilitic remedies, and have a strong tendency to relapses.

In ectropium luxurians MAXIMOWITSCH (431) recommends Peunow's method of operation—excising the projecting fold of conjunctiva and uniting the edges of the wound with sutures.

HIRSCHMANN.

TAMAMSCHEW (432) splits the lid as in the first step of Jaesche-Arlt's operation, and then cauterizes the wound with the nitrate-of-silver stick. He praises his results. HIRSCHMANN.

van Duyse (433) describes at length a case of unilateral congenital elephantiasis of the upper lid. Marckwort.

In operating for entropium de Vincentiis (434) splits the edge of the lid down to the tarsus, and then, without excising any skin, passes those loops of horse-hair from the conjunctiva through the edge of the tarsus and the skin of the lid, and then through the ciliary margin. By tying the loops the movable cilary margin is supported from above and the desired change in the direction of the lashes obtained. The horse-hairs are tied over a thin perforated plate of aluminium to prevent them from cutting through the thin skin of the margin. The gaping incision is left to cicatrization. The author considers the method especially adapted for those cases in which previous excisions of skin have been made without success, and the outer surface of the lid has therefore been shortened. Dantone.

VIII.—LACHRYMAL APPARATUS.

435. Gifford, H. Extirpation of the lachrymal gland causing atrophy of the optic nerve into the orbit. *Amer. Jour. Ophth.*, 1889, No. 9, p. 268.

436. Williams. Aluminium lachrymal probes and styles. *St. Louis Med. and Surg. Jour.*, 1889, No. 5.

IX.—MUSCLES AND NERVES.

437. Beard, C. H. Advancement with but a single suture. *Amer. Jour. Ophth.*, vol. vi., 3–4, p. 74.

438. Hansen–Grut. The pathogeny of divergent and convergent strabismus. *Trans. Ophth. Soc.*, 1888-89, vol. ix.

439. Leszynsky, M. Bilateral blepharospasm with divergent strabismus, cured after division of the external rectus muscle. Subsequent restoration of stereoscopic vision. *N. Y. Med. Jour.*, 1889, No. 20, p. 546.

440. Marlow. The position of rest as a cause of strabismus. *Ophth. Rev.*, vol. viii., p. 353.

441. Parinaud. Paralysie et contracture de la convergence, *Soc. d'opht. de Paris*, Dec. 3, 1889.

442. Pomeroy, O. D. Cases illustrating the influence of convex glasses in correcting the convergent squint of young children, mostly without tenotomy. *N. Y. Med. Jour.*, 1889, No. 16.

443. VICIANO. Ruptures traumatiques des muscles de l'œil. *Arch. d'opht.*, 1889, No. 6, p. 508.

444. VISSERING. On relapsing paralysis of the oculomotor nerve. *Münchener med. Wochenschr.*, 1889, No. 41.

BEARD (437) performs advancement by first incising the conjunctiva horizontally and laying the muscle bare. A thread with two needles is then prepared, one needle being passed through the muscle near the upper edge from above downward; the other in the same manner at the lower edge from below upward, after which the muscle is detached from the sclera. Next the needles are passed through the sclera near the corneal margin, after which one of them is passed through the loop from behind. By then drawing the muscle forward and tying the threads, the effect of the operation can be regulated with accuracy.

According to HANSEN-GRUT (438) the position of rest of the eyes is one of divergence, as when one eye is excluded from the visual act it generally deviates outward, as the orbital axes diverge, and as there is divergence in deep sleep and in narcosis. In ametropia there is a tendency to latent strabismus; in hypermetropia to convergent; in myopia to divergent strabismus. Convergent strabismus is the result of an innervation which produces a greater shortening of the recti than is desirable. If this abnormal innervation ceases, either permanently or temporarily, the strabismus disappears. There is no structural change in the muscles. Divergent strabismus is the result of diminished innervation of convergence, permitting one eye to assume its anatomical position of rest. Convergent strabismus in emmetropia is probably due to a change in refraction, there having been hypermetropia at the time of its development. No shortening of the muscles has ever been demonstrated as the cause. WERNER.

MARLOW (440) tested the position of rest in 20 cases of E., 71 of M., and 110 of H. In opposition to Stilling, he found a convergent position of rest about equally divided among the different refractive states. In myopes he found divergence in only 19 % in the position of rest, but in 40 % a vertical deviation.

PARINAUD (441) again discusses (*v. Arch. de Neurol.*, March, 1883) the paralysis and contraction of convergence. He distinguishes two kinds of paralysis of convergence: the combined and essential. The former is characterized by the fact that the paralysis of convergence is associated with paralysis of the upward and downward movement, while the lateral movements are not affected. In essential paralysis of convergence, on the contrary, only those muscles have ceased to act which play a part in fixing a near object : the muscles controlling convergence, accommodation, and contraction of the pupil. Parinaud describes another case of paralysis of convergence and accommodation, and two more of contraction of convergence and accommodation.

MAKCKWORT.

VICIANO (443) reports three new cases of rupture of the ocular muscles, the muscles affected being the levator palp. and the superior rectus.

VISSERING'S (444) patient was a boy aged fourteen, who had suffered with right-sided periodic headache since he was nine years old. The upper lid was always paralyzed. When the attack came there was nausea and increased salivation. Close observation of such an attack proved the affection to be an almost complete paralysis of the right oculomotor nerve. The fundus was normal, but the sensibility was diminished in the region innervated by the first and second branches of the fifth nerve, and there was marked salivation. At all other times every thing was normal. The patient gradually improved. Vissering seeks the cause of the affection in a sort of auto-intoxication, and compares it with megrim and epilepsy. Antipyrine was given with good results.

X.—ORBIT AND NEIGHBORING CAVITIES.

445. ALT, A. A fatal case of phlegmone of the orbit. *Am. Jour. Ophth.*, 1889, No. 10, p. 307.

446. ALT, A. Two cases of orbital sarcoma in children.

447. BULL, C. S. Contribution to the subject of tumors of the orbit and neighboring cavities. *N. Y. Med. Rec.*, Aug. 24, 1889.

448. EMRYS-JONES. Cavernous angioma of the orbit. *Trans. Ophth. Soc.*, 1888–89, vol. ix., p. 59.

449. KNAPP, H. The treatment of caries and necrosis of the orbit. *Amer. Ophth. Soc.*, 1889.

450. LANG, W. Traumatic enophthalmos with retention of perfect acuteness of vision. *Trans. Ophth. Soc.*, 1888–89, vol. ix., p. 41.

451. ROCKLIFFE, W. Deep suppurating hydatid of orbit. *Trans. Ophth. Soc.*, 1888–89, vol. ix., p. 55.

452. Sapieschko, K. M. Contribution to the question of the diseases of the frontal sinus. *Wjestnik Ophth.*, 1889, No. 6, p. 481.

453. Sharp. Case of hydatid of the orbit. *Brit. Med. Jour.*, 1889, No. 1,517.

454. Silcock, Quarry. Case of hyperostosis of the frontal bone and walls of the orbit, in which the morbid growth ceased to extend after removal of a portion of the diseased bone. *Trans. Ophth. Soc.*, 1888–89, vol. ix., p. 46.

Alt (446) operated on two children for sarcoma of the orbit. One died of erysipelas, in the other a relapse occurred.

In the case of Emrys-Jones (448) a tumor which did not pulsate filled the space between the external rectus and the globe. The course of the muscle over the tumor could be recognized. When the latter was removed there was only a slight hemorrhage, though it proved to be an angioma. Werner.

In treating caries and necrosis of the orbit Knapp (449) examines into the condition of the neighboring cavities. He opens and drains abscesses, scrapes rough parts of bone, and removes necrosed portions, as soon as they begin to yield. When the closure of the lids becomes imperfect he does not long postpone the operation, in order to prevent ulceration of the cornea.

A month after a boy, æt. thirteen, had received a blow upon the region of the eyebrows, which divided the lid and caused bleeding from the nose, Lang (450) observed a sinking back of the eye into the orbit, as though the patient were wearing an artificial eye. The movements of the globe were limited, the skin insensible, and smell impaired on the same side. $V = \frac{6}{5}$.

Werner.

Rockliffe's (451) patient, a man, æt. thirty-three, had suffered for six years with repeated inflammatory attacks, in consequence of which sight had been lost and proptosis had developed. During the operation a pus cavity was found at the apex of the orbit, which contained a hydatid cyst, but no hooks or no scolex were found. Werner.

Sapieschko (452), from his own observations and those of others, comes to the following conclusions in regard to the diseases of the frontal sinus: (1) dislocation of the eyeball downward and outward in young persons, or downward and slightly outward in adults; complete mobility of the eye may indicate dilatation of the frontal sinus of the corresponding side. Periodic plentiful mucous discharge from one nostril may be considered a confirmation of this diagnosis ; (2) headache, especially in the anterior part of the head and at the base of the nose, dizziness, and a sad frame of mind also add to the probability of the existence of such an affection ; (3) existence of a fistula near the inner lower orbital margin, with muco-purulent contents, with the presence of the above-mentioned symptoms, indicate empyema of the frontal sinus ; (4) pulsating discharge of the muco-purulent secretion of the fistula confirms the diagnosis and shows that it leads to a vascular cavity ; (5) to cure the affection it is necessary to trephine the anterior wall of the frontal sinus and establish free communication between it and the nasal cavity, which must be kept permanently open by drainage. HIRSCHMANN.

SILCOCK'S (454) case was one of hyperostosis of the frontal bone, which was removed from the frontal sinus. WERNER.

XI.—CONJUNCTIVA, CORNEA, SCLERA, ANTERIOR CHAMBER.

455. ADAMÜCK. Contribution to the knowledge of trachoma. *Wjestnik Ophth.*, 1889, No. 6, p. 505.

456. ALT, A. On pterygium. *Amer. Jour. Ophth.*, 1889, 3–4, p. 77.

457. DUPONT. Sur naphthol dans le traitement de quelques affections oculaires. *Thèse de Paris*, 1889.

458. GOMBERT. Recherches expérimentales sur les microbes des conjonctives à l'état normal. *Thèse de Montpellier*, 1889.

459. GRIFFITH, HILL. Case of primary lupus of the ocular conjunctiva. *Med. Chronicle*, Nov., 1889.

460. GRUENING, E. The use of the curette in trachomatous pannus. *Amer. Ophth. Soc.*, 1889.

461. GUAITA. Dosi piu convenienti e processo perferibile per l'applicazione del sublimato corrosivo e sua efficacia contro le varie forme di oftalmia granulosa. *Ann. di ottalm.*, vol. xviii., 4-5, p. 356.

462. GUNN, M. On a case of subconjunctival cysticercus. *Brit. Med. Jour.*, 1889, No. 1,486, p. 1405.

463. HOWE, L. Purulent conjunctivitis of infants and blindness in New York State. *Internat. Jour. Surg.*, vol. ii., No. 3, p. 54.

464. KNUS, T. Clinical and anatomical facts on vernal catarrh of the conjunctiva. *Inaug. Diss.* Zurich, 1889.

465. Malgat. Observations cliniques sur les granulations conjonctivales. *Rec. d'opht.*, 1889, No. 10, p. 577.

466. Norrie, G. Et tilfalde af primar tuberculosis conjunctiva. *Hospitalstidende*, 1889, No. 18.

467. Prince, A. E. Expression in the treatment of trachoma. *N. Y. Med. Jour.*, vol. l., 5, p. 138.

468. Puech. Traitement preventif de l'ophtalmie purulente des nouveau-nés. *Montpellier méd.*, 1889. p. 272.

469. Rampoldi. Storia di un doppio trapianto di conjunctiva e di cute, felicemente seguito per protesi oculare. *Ann. di ottalm.*, vol. xviii., 5–6, p. 368.

470. Reid, Thomas. On the pathology of trachoma and the relation of this condition to the other forms of conjunctival inflammation. *Ophth. Soc. Unit. Kingd.*, Oct. 17, 1889.

471. Werner, L. A case of subconjunctival cysticercus; microscopic examination of the enveloping capsule. *Trans. Ophth. Soc.*, 1888–1889, vol. ix., p. 74.

472. Williams. Defects in conjunctiva supplied from mucous membrane of mouth. *St. Louis Med. and Surg. Jour.*, vol. lvi., No. 9, p. 160.

473. Wolfe, J. R. On the transplantation of rabbit conjunctiva to the human eye. *Lancet*, Dec., 1889, p. 1219.

Adamück (455) again asserts that the white raised dots on the conjunctiva of the tarsus of the upper lids secure the diagnosis of trachoma, while their absence speaks against the existence of true trachoma. Hirschmann.

In granular conjunctivitis, Dupont (457) washes the inner surface of the lids with a solution of 0.2 : 1,000, and uses a naphthol salve (0.1 : 10 vaseline) for massage of the granulations. When nitrate of silver is used in blennorrhœa neonatorum, he nevertheless uses a solution of naphthol for washing the lids. He prefers naphthol α to β. Marckwort.

Gombert (458) finds that under normal conditions only a few microbes are found upon the healthy conjunctiva, as they are washed away by the tears as soon as they enter. The gonococcus and trachoma coccus make an exception, as they proliferate when they have once come in contact with the conjunctiva.

Marckwort.

In eleven cases of trachomatous pannus, Gruening (460) scraped off the pannus with a curette after the application of a strong solution of cocaine. In three cases the operation had to be repeated; otherwise the result was good.

GUAITA (461) again recommends the treatment of trachoma with sublimate, according to the following method: Daily brushing with a solution, 1 : 400, applied with a gauze brush under pressure, so that the remedy can penetrate into the fissures between the papillary excrescences. In milder cases the instillation twice a day of a solution, 1 : 1,000, is sufficient; in all cases the eyes should be washed several times a day with a solution, 1 : 5,000. DANTONE.

Based upon his observations of 64 cases of spring catarrh, 54 men and 10 women, KNUS (464) concludes that the most characteristic symptom of the disease is its regular yearly recurrence in spring and its disappearance in the cold months. The microscope revealed an enormous proliferation of epithelial cells. No micro-organisms could be found as the cause. He considers the affection a hyperplastic conjunctivitis.

MALGAT (465) believes that trachoma is transmitted in most cases by direct contact of the pus with the conjunctiva, though there are cases in which no infection has taken place. He thinks that the granulations are but one of the thousand forms in which scrofula manifests itself. Bad hygienic conditions, as is well known, play an important part in the development of trachoma. MARCKWORT.

In NORRIE'S (466) case no tubercle bacilli were found in the pus or in the scraped-out tissue; but inoculations of the anterior chamber of a rabbit gave positive results. SCHIÖTZ.

In a young woman who suffered with atrophy of the globe, and could not wear an artificial eye on account of the shrinkage of the conjunctiva, RAMPOLDI (469) transplanted a piece of rabbit conjunctiva, 15 *mm*, to the lower lid, and a piece of ordinary, skin 14 *mm* long, to the upper lid, taken from the upper arm of a young man. The result was very satisfactory. DANTONE.

REID (470) considers the appearance of groups of lymph cells in the subepithelial layer the principal symptom of trachoma. Secondarily, changes takes place in the epithelial layer, which gradually lead to the development of irregular papillary excrescences. Mucous follicles also form between the lymph follicles or in the centre of one of the latter. If they do not soften or are not absorbed, they may change to dense connective tissue, which forms the gray, cartilaginous granulations which are characteristic of the advanced stage of trachoma. Pathologico-anatomically follicular conjunctivitis is identical with trachoma, but it differs chemically, as it has not the tendency to form the firm cartilaginous nodules. The essential feature of purulent and catarrhal conjunctivitis consists in congestion of the blood, which leads to the accumulation of lymph and the formation of lymph-follicles, while trachoma and follicular conjunctivitis are an affection of the lymph-follicles.

In a boy seven years old, WERNER (471) found near the corneal margin a yellowish tumor, completely movable, under the con junctiva. It was excised and proved to be a cysticercus.

WERNER.

474. BAKER, A. R. Some remarks on transplantation of the cornea and allied subjects. *Amer. Jour. Ophth.*, No. 1, p. 1.

475. CASTALDI. Ulcus serpens ed hypopyon-keratitis. Napoli, 1889.

476. GRADENIGO. Del trapianto della cornea del pollo sull' occhio umano. *Boll. d'ocul.*, vol. xi., 17–18, p. 121.

477. HANSELL. On corneal abscess. *Amer. Ophth. Soc.*, 1889.

478. LAWFORD, J. R. Remarks on keratitis punctata, or descemetitis. *Ophth. Hosp. Rep.*, vol. xii., 4, p. 298.

479. LJUBINSKI, A. Keratitis dendritica exulcerans mycotica. *Wjestnik Ophth.*, 1889, No. 4, p. 345.

480. MILLINGEN, E. VAN. L'ozène et les ulcères infectieux de la cornée. *Arch d'opht.*, vol. ix., No. 6, p. 526.

481. PRICE, J. A. P. Congenital tumor of the left cornea. *Trans. Ophth. Soc.*, 1888–89, vol. ix., p. 168.

482. WILLIAMS. Removal of dermoid tumor from the cornea. *St. Louis Med. and Surg. Jour.*, vol. lvii., No. 5, p. 305.

BAKER (474) inserted a piece of glass, the shape of a collar-button, in place of an excised portion of leucomatous cornea. It healed in and was borne well for a while. After a few months the eye began to shrink, and one and one half years later the patient was totally blind.

CASTALDI (475) discusses at length the microbic nature of corneal ulcers, and strictly separates the serpent from the infectious ulcer, in respect to the cocci causing them, as well as to the appearance of the infiltrated tissue, and as to the prognosis. Infection of the cornea with staphylococcus aureus he considers a very severe disease; it does not cease with the perforation of the cornea, but goes on until the eye is destroyed. DANTONE.

GRADENIGO (476) succeeded in transplanting the cornea of a cock upon a human eye with total leucoma. The leucoma was made vascular a few weeks before the operation by detaching the conjunctiva from the sclera and drawing it over the cornea, where it was held with sutures. The transplantation was performed by removing the central part of the leucoma and inserting the whole cornea of the cock with the adjacent fibres of the sclera in its place, keeping it in place with four sutures and covering the insertion with the once more detached conjunctiva. Healing progressed favorably; the patient could distinguish large objects through the black pupil. On the twelfth day blood-vessels appeared on the new corneal margin, and on the twentieth exfoliation of epithelium began. Nothing is said in regard to the final result. DANTONE.

HANSELL (477) does not use cocaine in corneal abscess and other inflammatory affections of the cornea, on account of its destruction of the epithelium. He instils eserine and atropine alternately, and operates only when it is necessary to evacuate pus.

According to LAWFORD (478), the deposits on Descemet's membrane in descemetitis come from the uveal tract.

LJUBINSKI (479) observed, in 1884, in the right eye of Admiral Sch., and in 1887 in the left eye, a form of keratitis which closely resembles what has been described by Emmert as keratitis dendritica mycotica. In the right eye there was a superficial ramified opacity, projecting above the surface, which disappeared without a trace after a month, after passing into superficial ulceration. In the left eye the opacity lay deeper, returned after some improvement, led to extensive ulceration, was complicated with iritis, leaving synechiæ, and lasted four months. A circumscribed corneal opacity remained. In both eyes there were granulations upon the conjunctiva while the disease lasted, but disappeared upon its cure. There was no tuberculosis or other infectious disease. The infection must therefore have been an external accidental one. HIRSCHMANN.

MILLINGEN (480) calls attention to the pertinacity of ulcerous corneal affections complicated with ozæna, and thinks that it produces special forms of conjunctivitis and keratitis. He cites thirty-three cases of ocular affections complicated with ozæna, among them many diseases of the ear passages. v. MITTELSTÄDT.

483. Korjenewski. A case of bite of the sclera. *Medicinskoje Obosrenije*, 1889, No. 9.

484. Mitchell, J. Eserine in episcleritis. *Am. Jour. Ophth.*

485. Rockliffe, W. C. Case of sarcoma of the eyeball. *Trans. Ophth. Soc.*, 1888–89, vol. ix., p. 99.

486. de Vincentiis. Degenérazione jalina in uno stafiloma sclerale anteriore con glaucoma secondario. *Ann. di ottalm.*, vol. xviii., 4–5, p. 398.

Korjenewski (483) reports a case of dog-bite of the conjunctiva and sclera, with loss of substance (about 3 *mm* in diameter), which healed in four days. Hirschmann.

de Vincentiis (486) found, upon examination of an eye which had been enucleated on account of partial staphyloma and secondary glaucoma, a hyaline substance, which he ascribes partly to compression of an older fibrinous exudation, partly to direct deposit from the blood-vessels. The retina, which was absent, had probably also undergone hyaline degeneration.

Dantone.

487. Collins, W. J. Intrusion of eyelash into anterior chamber. *Trans. Ophth. Soc.*, 1888–89, vol. ix., p. 173.

Collins (487) successfully removed an eyelash from the anterior chamber forty-eight hours after the injury. Werner.

Sections XII.–XXI. Reviewed by Dr. P. SILEX.

XII.—IRIS.

488. Benson, H. A case of transparent iris cyst. With a colored illustration. *Trans. Ophth. Soc.*, vol. ix., p. 105.

489. Gunn, R. M. Peculiar congenital malformations of the eyeball affecting the iris, lens, vitreous, and fundus. *Trans. Ophth. Soc. Unit. Kingd.*, vol. ix., p. 166.

490. Lagrange, Felix. Du myome du corps ciliaire. *Arch. d'opht.*, vol. ix., No. 6, p. 484.

491. Rockliffe. Notes on a case of monocular suppurative (?) iritis. *Trans. Ophth. Soc.*, vol. ix., p. 99.

492. Sarkewitsch, N. A case of polycoria seu coloboma iridis multiplex. *Medicinskoje Obosrenije*, 1889, vol. xxxi., No. 5.

493. Straub, M. Bydrage tot de pathologische anatomie en physiologie der cyclitis. *Nederl. Tijdschrift voor Geneeskunde*, 1889, Deel ii., p. 806.

Benson's (488) patient, a man æt. twenty, presented a transparent cyst filling up the upper half of the anterior chamber. It was first noticed a year before the man came under observation, and was probably the result of an injury caused by a husk of oats. $V = \frac{6}{60}$. After removal of the cyst, which collapsed during the operation, the eye did well, and V rose to $\frac{6}{24}$ with a cylindrical glass. As in previously recorded cases, the inner surface of the cyst was lined with compound scaly epithelium. Werner.

In Gunn's (489) patient the sphincter portion of the iris was situated on a plane posterior to the outer circle, and tended to disappear under the latter when the pupil was dilated. Lens dislocated and notched, showing suspensory ligament. Opaque cord from lens to optic disc. Also appearances resembling opaque nerve fibres and coloboma of choroid, the latter above.

Werner.

Lagrange (490) describes the rare case of myoma of the ciliary body. It was observed in the right eye of a woman, æt. thirty-four, whose sight had been diminishing for the last two years. A roundish tumor, the size of a pea and color of raw meat, was visible downward and inward between the iris and lens, extending with its slightly transparent apex into the pupil. Behind the lens it merged into a second larger tumor with indistinct boundaries and of a bluish-white color, resembling detachment of the retina. The interior of the eye could not be clearly seen. On account of the uncertainty of the diagnosis the attempt was made to remove the tumor or cyst through a scleral section, but as sarcomatous masses then came to view, the whole eye was enucleated. The tumor consisted of three lobes, sat like a small nut upon the ciliary region, and included the lens downward and inward, the latter showing a deep indentation, but not being dislocated. The zonula at this point had disappeared, and there were only traces of the ciliary processes. Posteriorly the tumor was covered with the choroid, the anterior chamber and cornea were not affected, the retina was partly detached. The sclera, upon which the tumor rested, was normal. The tumor sprang from the ciliary muscle, and consisted mainly of muscular tissue, blood-vessels, and embryonic cells. It had developed mainly from the middle portion of the radial fibres, retaining the arrangement of the latter in its elements, while the meridional fibres did not participate. The latter, on the contrary, took the most active part in the development of the myoma described by Iwanoff. v. Mittelstädt.

Rockliffe (491) records a case of iritis in a child æt. eight. No family history of tuberculosis or syphilis. The surface of the iris was nodular, each nodule being yellow at the summit. The tension became increased, with ciliary staphyloma, later on — T, and the eyeball commenced to shrink. Enucleation. Microscopic appearances of inflammation only. **Werner.**

In the left eye of a soldier **Sarkewitsch** (492) found, besides a lateral pupil reacting to light and atropine, four other openings, which did not respond to light or atropine, of which the one to the nasal side of the pupil was twice the size of the latter. Both irides are irregularly pigmented. V of left eye $\frac{2}{40}$. Em. Fundus normal. **Hirschmann.**

Based on the anatomical examination of some cases of cyclitis, **Straub** (493) distinguishes between eyes with acute purulent cyclitis and eyes with so-called plastic cyclitis and slow course. In purulent cyclitis there is dense infiltration with leucocytes of the vascular part of the ciliary body, and a large purulent exudation invades the vitreous. A small portion passes through the fissures between the fibres of the ciliary muscle, and thus reaches the corner of the anterior chamber. In chronic cyclitis there is an exudation around the lens and in the anterior part of the vitreous. The perichoroidal fissures in the tissue are dilated and filled with coagulated albumen. In the vitreous there is a funnel-shaped or conical mass, with the ora serrata as its base and the papilla as its apex. This cone consists of compressed vitreous membranes. The outermost layer is the membrana limitans hyaloideæ. If the point of attachment to the papilla gives way, all the membranes withdraw and form what is known as the "membrane cyclitique" lying against the lens, which is not an exudation, but contains one. This exudation consists of leucocytes and newly formed blood-vessels. Assuming that the ciliary body secretes the lymph for the eye, and that in chronic cyclitis this part of the globe is diseased, as may be assumed from the marked diminution of tension and the occlusion of the ciliary blood-vessels with leucocytes, the hypothesis is justified that when the secretion of aqueous humor is impaired, liquid is abstracted from the vitreous, leading to the diminution of the volume of the latter with all the consequences described above. **Westhoff.**

XIII.—CHOROID.

494. **Dinkler, M.** Contribution to the knowledge of miliary tuberculosis of the choroid. *v. Graefe's Arch. f. Ophth.*, vol. xxxv., 4, p. 309.

495. Thompson, J. H. Peliosis rheumatica; suppurating irido-choroiditis in both eyes. *Kansas City Med. Jour.*, Nov., 1889.

The anatomical changes in Dinkler's (494) case consisted of small-celled, diffuse, and circumscript infiltrations. The latter showed microscopically the structure of the miliary tubercle. The ophthalmoscopic decoloration could be referred to changes in the pigment epithelium, obliteration of the blood-vessels, and necrosis of the tissue. The tubercles were remarkable on account of the formation of pigmented giant-cells and their varying development in the chorio-capillaris and the choroid proper. When they were in the latter they led to the destruction of the choroidal pigment, but when found in the former there was only a shifting of the pigmented connective-tissue cells. In the diffuse infiltration no tubercle bacilli were found, so that for this reason, as well as on account of its structure, it must be considered a secondary reactive kind of inflammation. Only the area supplied by the posterior ciliary arteries was affected.

Thompson (495) reports the history of a case of a woman who, while affected with peliosis rheumatica, was attacked with double purulent irido-choroiditis, which, in spite of all treatment, went on to the destruction of both eyes. Burnett.

XIV.—GLAUCOMA.

496. Castaldi. Nota clinica sopra un caso di glaucoma. *Ann. di ottalm.*, vol. xviii., 4–5, p. 417.

After Castaldi (496) had operated by iridectomy upon a case of acute glaucoma, which had been preceded for months by a slight degree of mydriasis, diminished tension, and stasis in the fundus, an acute attack developed in the other eye after the lapse of ten minutes, and an operation was at once performed. He ascribes the increase of pressure to the greater secretion into the vitreous in consequence of spasmodic contraction of the longitudinal muscular fibres of the blood-vessels. Dantone.

XV.—SYMPATHETIC OPHTHALMIA.

497. Ovio. Esame anatomo-pathologico di otto bulbi enucleati per ottalmia simpatica incipiente. *Ann. di ottalm.*, vol xviii., 4–5, p. 330.

498. Randolph, L. A contribution to the genesis of sympathetic ophthalmia; an experimental study. *Arch. f. Augenhk.*, vol. xxi., 2, p. 159.

Ovio (497) describes eight cases of sympathetic ophthalmia, in which the eye primarily affected was enucleated and carefully examined under the microscope. In none of them did he find any products of inflammation or parasitic germs at the optic nerve. Neither could any change be detected with the ophthalmoscope in any of the eight eyes in the optic nerve. He there considers the irritation of the ciliary nerves, which he found in all cases, the principal transmitter of the sympathetic affection.

Dantone.

XVI.—LENS.

499. Barban. Contribution à l'étude de l'extraction capsulaire partielle ou totale dans l'operation de la cataracte.

500. Berger, L. De l'extraction de la cataracte avec ou sans iridectomie. *Thèse de Lyon*, 1889.

501. Berry. De l'enclavement de l'iris consecutif à l'extraction de la cataracte, principalement d'après la méthode française. Historique, pathogénie, prophylaxie. Paris, Baillière, 1889.

502. Bull, C. S. Cataract extraction without iridectomy. *N. Y. Med. Jour.*, Nov. 2, 1889.

503. Collins, W. T. The composition of the human lens in health and in cataract. *Ophth. Rev.*, vol. viii., p. 321.

504. Doyne, R. W. Peculiar form of degeneration of the lens. *Trans. Ophth. Soc. Unit. Kingd.*, vol. ix., p. 113.

505. Gifford, H. A new dressing for cataract extraction and other wounds of the eyeball. These Arch., vol. xix., 1, p. 42.

506. McKeown, D. Clinical lecture on massage, scoops, and irrigation in the extraction of cataract. *Lancet*, Oct., 1889, pp. 783 and 992.

507. Steffan and Wecker. Correspondence on cataract operation. *Zeh. klin. Monatsbl.*, vol. xxvii., pp. 407 and 415.

508. de Vincentiis. Notizie intorno ad un inferma operata di cataratta oll' età di 107 anni. *Ann. di ottalm.*, vol xviii., 4–5, p. 181.

509. Webster. A fatal case of panophthalmitis from meningitis after cataract extraction. *Arch. f. Augenhk.*, vol. xxi., p. 191.

Barban (499) reports on forty-six cases of opening of the capsule with Wecker's capsule forceps made at Gayet's clinic. The results were very unsatisfactory, so that Gayet abandoned this method. Its dangers lie in injuries to the iris and cornea, prolapse of vitreous, incarcerations and adhesions, and intraocular hemorrhage. MARCKWORT.

BERRY'S (501) discussion of incarceration of the iris after cataract extraction is based upon experience at Gayet's clinic. Among 2,703 extractions *with* iridectomy, 1.3 to 2.1 % of incarcerations were observed; among 1,322 operations without iridectomy, incarceration occurred in 8.2 %, and in 1888, among 321 operations, even 41 incarcerations, *i.e.*, 13 %. MARCKWORT.

BULL'S (502) paper is based upon one hundred cases of extraction without iridectomy, which are tabulated carefully. He uses a narrow Graefe knife, and includes the upper two fifths of the cornea in the section. A *V* or square incision is then made in the capsule, and the lens pressed out with a rubber spoon applied to the lower edge of the cornea. He washes out the anterior chamber with Panas' fluid and instils a solution of eserine, half a grain to the ounce. Both eyes are closed and a roller bandage is applied. On the whole, he thinks the advantages of the operation are greater than its disadvantages. BURNETT.

COLLINS (503) determined the weight, amount of total solids, water, and ash in six clear human lenses and ten cataractous. The weight of the clear lenses was found to increase with age, as previously shown by Priestley Smith, but the average amount of water (70 %) and solids (30 %) remains the same. In no case did the weight of a cataractous lens approach the weight of even the lightest clear lens, but the ratio of water (65 %) to solids (35 %) was increased in cataract. As far as chemical changes are concerned, the author believes that the cataractous process is not an exaggerated senile change, but due to disturbance of nutrition of another nature. WERNER.

DOYNE (504) records an instance of a sharply defined difference of 11 D between the nucleus (— 3 D) and the cortex (— 14 D) of the lens. A nuclear opacity was visible by reflected light, but not by transmitted light. $V = \frac{6}{36}$ with — 3 D; it began first to decrease and then rose again until, with — 14 D, $V = \frac{6}{36}$.

WERNER.

MCKEOWN (506) gives a short account of the history of the scoop in cataract extraction, alludes to the difficulty of diagnosing the ripeness of a cataract, and describes a new siphon irrigator for removing cortical remains. According to his more recent experience, he has had less iritis than formerly; vitreous was lost only once in 1888, and not at all in 1889. In 177 cases he had four suppurations, which he believes were not attributable to the irrigation. WERNER.

STEFFAN (507) still maintains that technique is of the greatest importance in obtaining good results. Strict cleanliness accomplishes as much in ophthalmic surgery as antisepsis, which cannot be thoroughly carried out. WECKER considers antisepsis absolutely necessary for the attainment of good results. The incision must be normal, otherwise incarceration and subsequent infection may follow. Of course he argues in favor of the round pupil. He considers a preliminary iridectomy a great step backward.

DE VINCENTIIS (508) performed an extraction with a small flap, according to Wecker, on a woman one hundred and seven years old, with excellent result. DANTONE.

XVII.—VITREOUS.

510. MAYWEG. On recurrent hemorrhage into the vitreous. Report of the Ophth. Soc. at Heidelberg. *Arch. f. Augenhk.*, vol. xxi., 1, p. 104.

511. WAGEMANN. On the purulent infiltration of the vitreous from operative scars and synechiæ. Report of the Ophth. Soc. at Heidelberg. 1889. *Arch. f. Augenhk.*, vol. xxi., 1, p. 102 ; and *Graefe's Arch. f. Ophth.*, vol. xxxv., 4, p. 116.

XVIII.—RETINA AND FUNCTIONAL DISTURBANCES.

512. ABADIE, CH. Traitement du décollement de la rétine. *Ann. d'ocul.*, vol. cii., p. 203.

513. BAKER, CH. R. Impaired vision as the result of sunstroke. *Jour. Am. Med. Assoc.*, Dec. 7, 1889.

514. DOLARD, L. Des altérations pigmentaires de la rétine consécutives à la phthisie oculaire. *Arch. d'opht.*, 1889, vol. ix. No. 6, p. 493, Nov.–Dec.

515. DOYNE. Recurrent transient blindness. *Trans. Ophth. Soc. Unit. Kingd.*, vol. ix., p. 148.

516. DUBOIS and RENAUT. Sur la continuité de l'epithélium pigmenté de la rétine avec les segments externes des cônes et des batonnets et la valeur morphologique de cette disposition chez les vertébrés. *Note présentée à l'Acad. des Sci.*, 1889.

517. FROST, A. Notes of a lecture on some forms of retinal pigmentation. *Brit. Med. Jour.*, Dec., 1889, p. 1319.

518. MITCHELL, J. K., and DE SCHWEINITZ. Hysterical anæsthesia with examination of the visual field. *Amer. Jour. Med. Sci.*, Nov., 1889.

519. MULES. Primary retinal phlebitis. *Trans. Ophth. Soc. Unit. Kingd.*, vol. ix., p. 130.

520. SCHÖLER. Brief remarks on the influence of tincture of iodine, in connection with the fatal case of detachment of the retina reported by Dr. Gelpke, which was treated according to this method. *Centrabl. f. prakt. Augenhk.*, vol. xiii., p. 327.

In detachment of the retina, ABADIE (512) does not inject tincture of iodine, as Schöler does, but 1 to 1½ drops of the following solution: tinct. iodi 5.0, aq. distill. 5.0, potass. iodid. 0.25. He makes a puncture with Graefe's knife at the point of detachment of the retina, permits the subretinal liquid to escape by turning the knife, and then injects the above-mentioned solution. MARCKWORT.

BAKER (513) has examined 13 cases where sun-stroke was an alleged cause of impaired vision, and he is of the belief that injury to sight is undoubtedly the result of sun-stroke in quite a large number of instances, generally in the form of atrophy of the nerve, with contraction of the V F.

BURNETT.

DOLARD (514) describes a pigment degeneration resembling that of true retinitis pigmentosa, which he saw in two eyes removed at Gayet's clinic, on account of total anterior cicatricial staphyloma. Even microscopically the pigment seemed generally confined to the blood-vessels, which appeared like black lines upon the bright background; at other points the pigment formed a delicate network, corresponding to the distribution of the smaller blood-vessels, but here and there it also was found scattered about the retina without any blood-vessels, not even obliterated ones, near it. The microscope revealed a conversion of the retina into connective tissue, with disappearance of the nervous elements, the rods and cones, and the pigment-epithelium. The papilla was excavated, the choroid normal. Gayet thinks that in such eyes there takes place with the destruction of the nervous elements a disintegration of the pigment-epithelium, the granules of which enter the retina along the sheaths of the blood-vessels and the capillaries. The above-mentioned changes are the same as those observed by various authors after accidental or experimental injuries to the optic nerve; they are the result of the loss of function and vitality of the retina, the sclerous degeneration of which follows the pigment-infiltration. v. Mittelstädt.

Doyne (515) records an instance in which transient blindness occurred twice in the same eye of a man, æt. 22, who had suffered from fever in India. The whole upper half of the retina was cloudy ; no change in the vessels. Field defective below. $V = \frac{6}{6}$. Five months later the field was unaltered ; upper half of disc white, upper arteries contracted on the disc. Berry believed that many cases of recurrent blindness were due to spasmodic constriction of the retinal vessels. Werner.

From their anatomical examinations, Dubois and Renaut (516) conclude that the rods and cones of the retina are not merely in simple contact with the pigment-epithelium, but closely united with it. This union, however, is easily dissolved in the dead eye. Marckwort.

Frost's (517) first figure illustrates a case of physiological pigmentation of the retina, occurring in the form of dots, as in Fig. 76 of Wicker and Jaeger's Atlas. The three other figures represent cases of retinitis pigmentosa, in two of which the clinical symptoms are typical, but there is marked atrophy of the choroid, and comparatively insignificant pigmentation. The author is inclined to believe that the pigment diminishes as the disease progresses. Werner.

Mitchell and de Schweinitz (518) give the histories in detail of eight cases of hysteria, taken at random from their practice. In no case was there a complete loss of color-sense, nor any disappearance of the colors in a constant order. In two cases the cutaneous anæsthesia was complete, but there was no departure from the normal sequence of the colors. In two cases the normal order of the limits of the color-fields was partially reversed ; in one case it was completely reversed. In none was any marked amblyopia present. It is interesting to note that these results are somewhat different from those found by European observers.

Burnett.

Mules (519) records three cases of what he calls primary phlebitis of the retina ; in two of these, thrombi were visible in the retinal veins. There was a central scotoma, with normal peripheral field. Hemorrhages not constant. No optic neuritis.

Werner.

Schöler (520) declares that tincture of iodine in the quantity injected cannot endanger life, either by toxic action, or by the formation of thrombi in the veins, or by direct transmission through the lymph-spaces of the eye into the liquor cerebrospinalis. In Gelpke's case, purulent choroiditis, with secondary meningitis, resulted from infection.

XIX.—OPTIC NERVE.

521. MANARA. Nevrite ottica retrobulbare. *Riforma med.*, June, 1889.

522. THOMPSON, T. L. Some cases of inflammation and atrophy of the optic nerve, with special reference to etiology and prognosis. *Jour. Am. Med. Assoc.*, Dec. 28, 1889.

MANARA (521) records a case of sudden and complete blindness in both eyes, of a poorly nourished driver, thirty-four years of age. The ophthalmoscope revealed œdema of the papilla and moderate tortuosity of the veins. As there were no symptoms indicating uræmia, exudation in the meninges, or brain-tumor, Professor Businelli made the diagnosis of simple, retrobulbar, rheumatic neuritis, with relatively good prognosis. After a month of derivative treatment, vision was completely restored, quantitative perception of light having returned on the sixth day after he became blind. The ophthalmoscopic changes had entirely disappeared when the patient was discharged. DANTONE.

THOMPSON (522) gives the histories of nine cases which bear upon the subject of optic-nerve inflammation and atrophy. His general conclusions are as follows : progressive atrophy, with no signs of previous inflammation, rarely give any clue to etiology, and the prognosis usually is not good. The atrophies following pernicious intermittent fevers sometimes unexpectedly improve. Neuritis, from meningitis of syphilitic origin, offers a good prognosis ; neuritis from non-syphilitic meningitis does not. Neuritis from brain-tumors is often variable, but death usually results. Neuritis from Bright's disease is always followed by death within two years. Neuritis attending alcohol and tobacco poisoning is always associated with permanent amblyopia, but when treated before atrophy sets in the prognosis is good. BURNETT.

XX.—INJURIES, FOREIGN BODIES (PARASITES).

523. HUTCHINSON, J. A form of retino-choroiditis due to concussion of the eye. *Trans. Ophth. Soc. Unit. Kingd.*, vol. ix., p. 116.

524. **Stölting.** Contribution to the knowledge of extraction of intraocular cysticerci. *Berliner klin. Wochenschr.*, 1889, No. 42.

525. **de Vincentiis.** Nuove osservazioni sui cisticerchi intra- et extra-oculari. *Ann. di ottalm.*, vol. xviii., 4–5, p. 382.

Hutchinson (523) records a case of pigmentary change in the macular region after a non-penetrating injury. In the author's opinion, such pigmentation is usually central and independent of hemorrhages. **Werner.**

Stölting (524) successfully extracted a subretinal cysticercus, which had developed near the optic nerve. Vision ten weeks after the operation equalled $\frac{4}{5}$ of the normal. By inserting needles into the globe and subsequent ophthalmoscopic examination, the pupil being dilated, he tried to locate the parasite accurately.

de Vincentiis (525) discusses the fourteen cysticerci which he observed, partly at Palermo, partly at Naples, and gives the clinical history of five cases latterly examined. Of these, two were subconjunctival, the others intraocular. Two of the latter, he thinks, were undoubtedly in the vitreous. **Dantone.**

XXI.—OCULAR AFFECTIONS IN CONSTITUTIONAL DISEASES.

526. **Bernhardt.** Contribution to the knowledge of the nuclear paralysis of the ocular muscles and recurrent paralysis of the oculomotor and facial nerves. *Berl. klin. Wochenschr.*, 1889, No. 47.

527. **Edmunds** and **Lawford.** Retinal changes in a fatal case of alcoholism. *Trans. Ophth. Soc. Unit. Kingd.*, vol. ix., p. 137.

528. **Friedenwald, A.** Disturbed equilibrium of the muscles of the eye, as a factor in the causation of nervous diseases. *Trans. Med. Chir. Soc., Faculty of Maryland*, 1889.

529. **Oppenheim.** Contribution to the knowledge of the syphilitic affections of the central nervous system. *Berliner klin. Wochenschr.*, 1889, No. 48.

530. **Oulmont.** Cécité subite par ramollissement des deux lobes occipitaux. *Gaz. hebd.*, 1889, No. 38, p. 607.

531. **Samelsohn.** On hemichromatopsia. *Berliner klin. Wochenschr.*, 1890, No. 13.

532. **Sattler.** On the relation of the eye to the constitutional affections of the body. *Allg. med. Centralztg.*, 1889, No. 100.

533. SCIMENI. Sulla nevrite ottica di tumor cerebrale. *Ann. di ottalm.*, vol. xviii., 4–5, p. 432.

534. WILLIAMS, HENRY W. Advances in our knowledge of some cerebral, ocular, and intraorbital lesions, which facilitate the diagnosis and treatment of important diseases. *Jour. Amer. Med. Assoc.*, Nov. 16, 1889.

BERNHARDT (526) reports a case of nuclear paralysis of the ocular muscles (abducent, internal rectus), two cases of recurrent oculomotor, and five of recurrent facial paralysis. He will not undertake to decide whether they were pure periodic or periodically exacerbating paralyses in Senator's sense. The paralyses of the facial nerve, however, were also sometimes observed on the other side. The purely periodic ones were accepted in accordance with Vissering as due to auto-intoxication, which at certain intervals shows toxic effects, while in the periodically exacerbating paralyses some anatomical change (tuberculous process, tumors, affections of the middle ear) is probably the cause.

EDMUNDS and LAWFORD (527) record a case of alcoholic paralysis with cloudiness of the whole retina and enlarged but not tortuous retinal veins. No syphilis. On examination after death the outer nuclear layer contained large spaces filled with non-fibrinous coagulum, between the fibres of Müller. Optic nerves normal. No central scotoma existed. WERNER.

FRIEDENWALD (528) gives no cases, but only considers the subject in a general way, expressing his belief in the causation of certain nervous diseases and general disorders by disturbed equilibrium of the external eye-muscles and in their cure by the Stevens method. BURNETT.

As the investigations of OPPENHEIM (529) and many others show, cerebral syphilis generally begins in the meninges. Diffuse broad and flat formation of new tissue, especially at the base, is more common than the development of well-defined tumors: meningitis diffusa basilaris syphilitica. The base appears filled out with a solidifying fluid, like paraffine. The large arteries are affected. The optic tracts and oculomotor nerves are frequently enclosed in a large tumor-like mass. Circumscribed softening and hemorrhages are generally found in the brain. The anatomical substratum consists of a vascular granulation-tissue rich in cells, the blood-vessels of which are partly closed by thrombi and obliterated; it shows amyloid and fibrous degeneration. The loose tissue around the chiasm is most frequently affected. The nerve-sheath becomes infiltrated and numerous offsets enter the nerve fibres. The latter become atrophic, while the nerve appears swollen. The oculomotor and optic nerves are often attacked; the affection of the latter especially can be easily traced through the stages of neuritis, choked disc, and neuritic atrophy. The visual disturbance very frequently indicates disease of the chiasma and tractus. The fleeting nature is almost pathognomonic for the visual disturbances and the paralyses of the ocular muscles. The second part of the article refers to spinal syphilis, which, though instructive, offers little that is interesting from an ophthalmoscopic point of view.

OULMONT (530) records five cases of blindness following destruction of the cortex of the occipital lobe, compiled by Chauffard, and adds one observed by himself, in which the focus of degeneration was a very extensive one. He concludes that total blindness can be produced by a lesion of both occipital lobes without any change in the optic nerve. MARCKWORT.

SAMELSOHN'S (531) case was one of the development of a semilateral defect for colors only. The autopsy showed that the theory that the location of the defect could only be in the cortex was false, a gliosarcoma of the optic tractus with participation of the optic thalamus and corpora quadrigemina being found. The cortex and visual-fibre radiation were not involved.

SCIMENI (533) anatomically examined five cases of cerebral tumors and found that the attending optic neuritis is of an inflammatory nature from the beginning, and only when ascending leads to further changes in the nerve. He saw hyperæmia of the retinal blood-vessels, exudation and formation of new vessels in the papilla, changes in the walls, etc., at a time when vascular alterations resulting from pressure by the tumor could be demonstrated in the intracranial, but not in the orbital, part of the nerve. Of the experiments made by the author on rabbits the injections of agar and mercury, alone or mixed, into the sheath of the optic nerve are the most noteworthy. A neuritis sometimes develops on the second day after the injection, just like the one observed in cerebral tumors. DANTONE.

WILLIAMS (534) in his paper gives some general considerations and thoughts derived from his own observations, with citation of cases on "Intracranial growths," "Intraocular conditions having important relations with other diseases," and "Phlegmonous inflammation of the orbital tissues as a sequel of facial erysipelas." Two interesting cases of abscess of the orbit are related in full.

BURNETT.

FIRST CUBAN MEDICAL CONGRESS HELD IN HAVANA FROM JANUARY 15 TO 22, 1890.

(From the *Revista de Ciencias med. de la Habana.*)

FIFTH SESSION.

DR. SANTOS FERNANDEZ : Diagnosis of Cataract.—It is not always easy to make the diagnosis of cataract ; for although in some cases this condition may be recognized or suspected at a distance from the aspect of the patient, in others it is not possible to arrive at a correct conclusion, even when close to the patient.

In a series of patients who were to have been operated on in succession, on arriving at the fourth it was noticed that the diagnosis was not clear, and later on a detachment of the retina was recognized. In another patient, in whom the diagnosis of cataract had been made abroad, the same condition was found. In both these cases the real condition was recognized by means of the ophthalmoscope ; so that when the mistake has been made in persons somewhat advanced in years it must be attributed to the non-employment of this means of diagnosis.

There have been some cases of error arising from deposits of iris pigment on the lens capsule. There have also been, according to some authorities, mistakes arising from opacities of the vitreous.

The ophthalmoscope ought to decide whether the patient should or should not be operated on.

There are some reported cases of spontaneous cure of cataract. A monk, who was blind, fell down a flight of stairs, and on picking himself up was agreeably surprised to find that he had recovered his sight. Another person who had cataract received a blow on the head and recovered his lost sight, and on his bending forward the opaque lens came back into its normal position, the person remaining in the same condition as before the blow.

There are some cases of doubtful diagnosis ; as happens in those where the lens has an ebony color and the pupil appears dark.

When cataract and glaucoma co-exist an iridectomy should first be performed, and later on the extraction of the lens.

Medicinal treatment has never been of use in dealing with incipient cataract.

It is not possible to fix the time necessary for the full development of a cataract. He asks whether that cataract which commences in the centre of the lens is that which takes the longest time in becoming mature ?

Dr. Santos Fernandez finished by stating that, notwithstanding the progress in the diagnosis of cataract from Purkinje (1823) to Helmholtz (1851), there was always a doubt before the latter's time as to whether there was an opacity of the lens or not.

Dr. Lopez believes the employment of the ophthalmoscope necessary in order to complete the diagnosis of cataract. He operates on both eyes in one sitting.

Dr. Santos Fernandez thinks that since the introduction of antisepsis a cataract may be operated on before it is mature. Operating on both eyes in one sitting is objectionable.

SIXTH SESSION.

Dr. Lopez : Treatment of Infected Corneal Ulcers with the Thermo-Cautery.—The author believes Saemisch's operation dangerous, difficult to perform, and not always successful in accomplishing the desired end ; whilst by the destruction of the infectious germs with red heat, as applied by the thermo-cautery, one obtains a simple ulcer, which heals rapidly with ordinary attention to cleanliness.

Dr. Montalvo has obtained good results with the use of a solution of bichloride of mercury of the strength of 1:2000.

Dr. Santos Fernandez believes that the result obtained in the treatment of these ulcers depends on the degree of infection.

NINTH SESSION.

Dr. Madan : Conjunctivitis in Arterial Sclerosis.—He describes several cases of conjunctivitis occurring in Bright's disease, which inflammation, together with other ocular disturbances, appeared long before the renal. He considers this fact of great diagnostic value, and one which ought to be added to the other ocular phenomena occurring in that disease.

Dr. Lopez relates the case of a patient who consulted him for a recurrent conjunctival œdema, in which the ophthalmoscope revealed Bright's retinitis.

Dr. Santos Fernandez has observed cases similar to that mentioned by Dr. Lopez.

MISCELLANEOUS NOTES.

Our reviewer and first assistant at the Ophthalmic Clinic of the University of Berlin, **Dr. Paul Silex**, has established himself as "Privatdocent" of Ophthalmology in that city.

Dr. Just died at Zittau, Jan. 5, 1890.

Dr. Jacob H. Hock died at Vienna, Feb. 2, 1890.

Dr. Struve, of Bremen, died suddenly at Wiesbaden, whither he had gone on account of his health.

Dr. L. F. Love has been appointed Ophthalmic Surgeon to St. Mary's Hospital, Philadelphia.

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